A study on the effect of massive pleural effusion on cardiovascular hemodynamics

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CHENNAI, INDIA

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CERTIFICATE

This is to certify that the dissertation titled **"A study on the effect of massive pleural effusion on cardiovascular hemodynamics"** is the bonafide original work of **Dr. MURUGA BHARATHY K.,** in partial fulfilment of the requirements for M.D. Branch–I (General Medicine) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in April 2012. The Period of study was from April 2011 to November 2011.

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DECLARATION

I hereby solemnly declare that the dissertation titled "**A study on the effect of massive pleural effusion on cardiovascular hemodynamics"** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3, during April 2011 to November 2011 under the guidance and supervision of my unit Chief Prof. C. RAJENDIRAN, M.D.

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of requirement for the award of M.D Degree (Branch-1) in General Medicine.

SIGNATURE OF THE CANDIDATE

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Date:

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ABBREVIATIONS

RA	right atrium
RV	right ventricle
LA	left ventricle
RVDC	Right ventricular diastolic collapse
PR	pulse rate
SYS BP	systolic blood pressure
DIA BP	diastolic blood pressure
RA cm	right atrial size in centimetre
LA cm	left atrial size in centimetre
RV basal	right ventricular dimension at the base
RV mid	right ventricular size at mid-level
RV base-apex	right ventricular size from base to apex
LV sys	left ventricular size in systole
LV dias	left ventricular size in diastole
SpO_2	pulse oximetry haemoglobin saturation
BTC	before thoracentesis
ATC	after thoracentesis
AV	Atrio-ventricular

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INTRODUCTION

Pleural effusion is a medical emergency if it is more than moderate. Pleural effusion of any etiology may compromise pulmonary function resulting in dyspnea. The close relation between pleura and pericardium and the dependence of their pressure kinetics are well known factors. Many literatures on pleural effusion deal with the effect of pleural effusion on gaseous functions and diaphragmatic functions[1, 2, 3, 4]and literatures are very few on the effects of pleural effusion on cardiovascular hemodynamics[5]. So a study on the impact of pleural effusion on cardiovascular hemodynamics taking into consideration all possible variables will bring into limelight the relationship between these two cavities and help to formulate concepts that may provide some insight into the pathophysiology of symptoms in pleural effusion.

The mediastinal pleura and the pericardium are closely related[6].In pleural effusion there is a gradient in the pleural pressure due to the hydrostatic column of fluid[6,7]. Accordingly, the pleural pressure in the dependent part of the hemithorax is much greater than that in the superior part of the hemithorax. So the collections in the pleural cavity can compromise cardiac function mimicking a tamponade. There are few case reports on cardiac chamber collapse in cases of massive pleural effusion[8,9]. Intra thoracic pressure is transmitted to cardiac chambers through pericardium, and one of the important determinants of intrathoracic pressure is intrapleural pressure. Intra pericardial pressure both approximates and varies with pleural pressure. The inspiratory decrement in

pleural pressure normally reduces pericardial pressure, RA, RV, pulmonary capillary wedge pressure and systemic arterial pressure slightly.

It has been shown in a canine model that large bilateral pleural effusions lead to an increase in intrapleural pressure[8], which causes a linear increase in intrapericardial pressure finally leading to right ventricular diastolic collapse[10, 11]. So cardiac tamponade physiology is not only related to pericardial effusions. Large pleural effusions of various etiologies, in the absence of significant pericardial effusions, have been reported to lead to cardiac tamponade physiology. Clinical manifestations of large pleural effusions include hemodynamic instability and pulsus paradoxus. Larger effusions and bilateral effusions cause higher incidence of ventricular collapse and hemodynamic instability.

AIMS AND OBJECTIVES

- To study the impact of pleural effusion on cardiovascular hemodynamics.
- To analyse whether the sidedness of pleural effusion has any impact on cardiovascular hemodynamics.
- To study the cardiovascular hemodynamics after therapeutic intervention of pleural effusion.

REVIEW OF LITERATURE

The pleura is a thin serous layer, which covers the lungs (visceral pleura) and is reflected, by way of the lung hila, on to the chest wall and pericardium (parietal pleura).The pleural space thus created extends from the root of the neck, 3 cm above the mid-point of the clavicle, down behind the abdominal cavity, in the costo-diaphragmatic recess, to the 12th rib overlying the kidney[12].Only a thin layer of pleural fluid separates the parietal and visceral pleura. The parietal layer secretes 2400mlof fluid daily, which is resorbed by the visceral layer[13].Pathological collection of fluid in pleural cavity leads to pleural effusion. Pleural pressure increases with accumulation of fluid. When pleural fluid is present, its volume must be compensated for by an increase in the size of the thoracic cavity, a decrease in the size of the lung or a decrease in the size of the heart, or a combination of these changes[6].

Since the thoracic cavity, lungs, and heart are all distensible objects, the volume of each is dependent on the pressure inside minus the pressure outside. The presence of pleural fluid increases the pleural pressure. Since the distending pressure of the thoracic wall is the atmospheric pressure minus the pleural pressure, an increase in the pleural pressure will lead to an increase in the distending pressure of the thoracic cavity and an increase in the volume of the thoracic cavity. The distending pressure of the lungs is the alveolar pressure minus the pleural pressure. Therefore, an increase in the pleural pressure will lead to a decreased lung volume. Since the distending pressure of the heart is the

intracardiac pressure minus the pleural pressure, an increase in the pleural pressure will lead to a decrease in the size of the heart[6].

Figure 1 Relationship between pleura and pericardium[14]

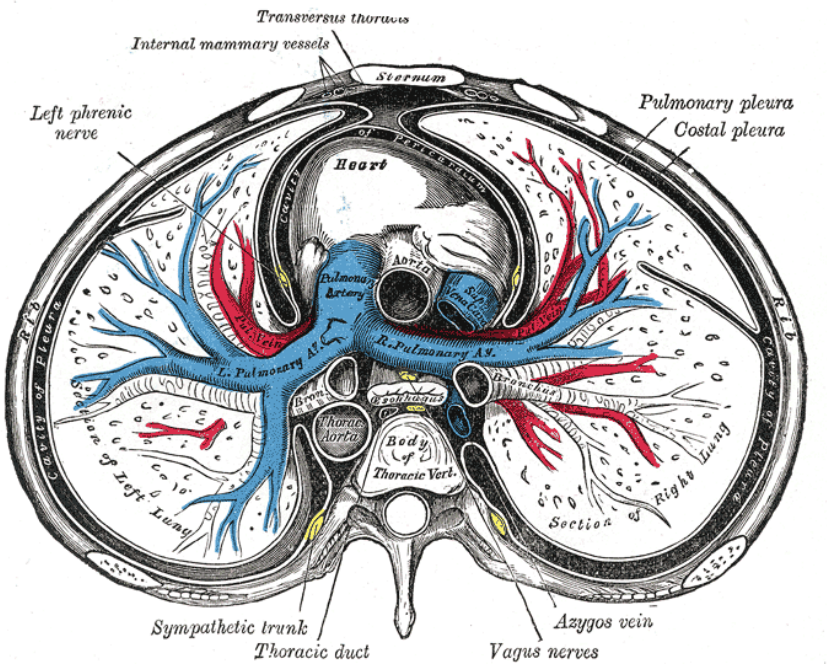
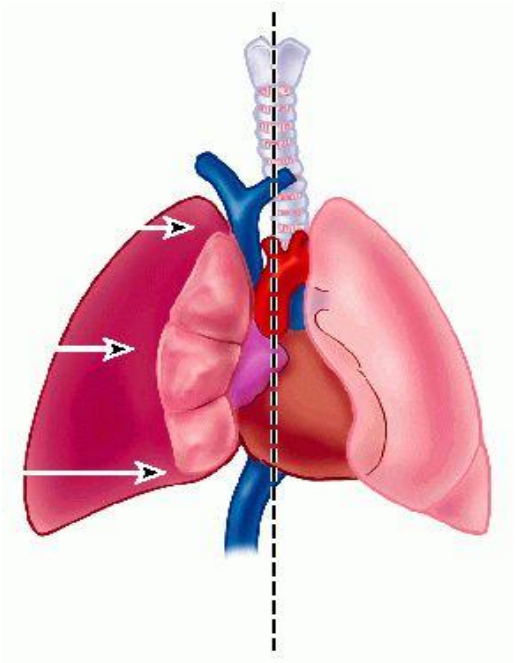


Figure 2 showing the effect of massive pleural effusion on heart



Pericardial space anatomy:

The pericardium is a fibroserous sac[15] that encloses the heart and the root of the great vessels. Its function is to restrict excessive movements of the heart as a whole and to serve as a lubricated container in which the different parts of the heart can contract.

The serous pericardium lines the fibrous pericardium and coats the heart. It is divided into parietal and visceral layers. The parietal layer lines the fibrous pericardium and is reflected around the roots of the great vessels to become continuous with the visceral layer of serous pericardium that closely covers the heart.

The visceral layer is closely applied to the heart and is often called the epicardium. The slit like space between the parietal and visceral layers is referred to as the pericardial cavity. Normally, the cavity contains a small amount of fluid (about 50 mL), the pericardial fluid, which acts as a lubricant to facilitate movements of the heart.

Pleural fluid physiology:

The pleural space contains a tiny amount (≈0.3 ml/kg) of hypo-oncotic fluid (≈1 g/dl protein). Pleural fluid turnover is estimated to be ≈0.15 ml/kg/h. Pleural fluid is produced at parietal pleural level, mainly in the less dependent regions of the cavity. Reabsorption is accomplished by parietal pleural lymphatics in the most dependent part of the cavity, on the diaphragmatic surface and in the mediastinal regions. The flow rate in pleural lymphatics can

increase in response to an increase in pleural fluid filtration, acting as a negative feedback mechanism to control pleural liquid volume. Such control is very efficient, as a 10 fold increase in filtration rate would only result in a 15% increase in pleural fluid volume. When filtration exceeds maximum pleural lymphatic flow, pleural effusion occurs[16].

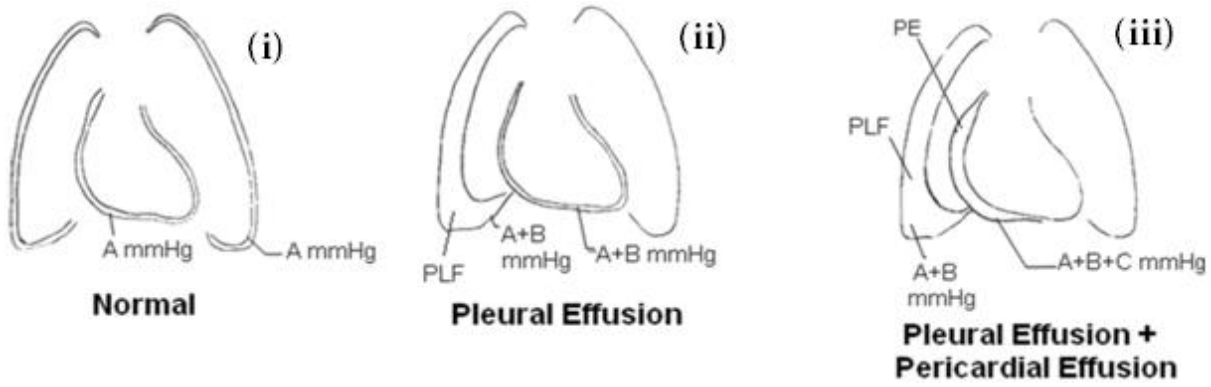
Relationship between pleura and pericardium:

The parietal pleura has costal, mediastinal, and diaphragmatic parts and a cupola. The costal pleura is separated from the sternum, costal cartilages, ribs, and muscles by a loose connective tissue termed endothoracic fascia, which provides a natural cleavage plane for surgical separation of the pleura from the thoracic wall. Anteriorly, the costal pleura turns sharply onto the mediastinum, and the underlying portion of the pleural cavity is called the costomediastinal recess. Inferiorly, the costal pleura is continuous with the diaphragmatic pleura, and the underlying space is termed the costodiaphragmatic recess. In the adult, the anterior borders of the right and left pleurae probably meet at or near the median plane during a part of their course. The left anterior border sometimes diverges to leave a part of the pericardium uncovered (bare area). Posteriorly, the pleura crosses the twelfth rib. At the root of the lung, the mediastinal pleura turns laterally, enclosing the structures at the root and becoming continuous with the visceral pleura. This reflection projects downward as a tapering double fold called the pulmonary ligament. The mediastinal pleura is adherent to the pericardium except where the phrenic nerve descends between them[17]. This

relationship between mediastinal pleura and pericardium is responsible for transmission of pressure alterations in the pleural cavity to pericardium and chambers of heart.

Mechanism of cardiac tamponade with a large pleural effusion:

Figure 3 Mechanism of cardiac tamponade in massive pleural effusion



The figure. 3 (i)[18] demonstrates a normal subject where intrapericardial pressure (A mmHg) and intrapleural pressures (A mmHg) are the same. The se figure. 3(ii) demonstrates a model where a pleural effusion is present and the intrapleural pressure increases from A to A+B. The pericardial pressure is also increased to A+B because intrapleural pressure is transmitted to the pericardial sac. If there is a concomitant pericardial effusion in addition to pleural effusion, the intrapericardial pressure will increase further to A+B+C (figure. 3(iii)).

PLF - pleural fluid and PE - pericardial effusion.

Pathophysiology of cardiac tamponade:

For each cardiac chamber, its transmural pressure ie. intracardiac pressure minus pericardial pressure, is a principal determinant of its filling(Transmural pressure is a true filling [distending] pressure that contributes to ventricular

preload.[19,20]).Normal pericardial pressure is lower than the right atrial mean and right ventricular diastolic pressure that right atrial transmural pressure (right atrial pressure minus pericardial pressure) is normally higher than its cavitary pressure. In tamponade, rising pericardial pressure progressively reduces the transmural atrial and ventricular pressure and ultimately can make the average transmural pressure of first the right and subsequently the left cardiac chambers phasically negative.[21,22] Survival necessitates the ensuing parallel rise in diastolic pressures, first in the right side of the heart and later the left side of the heart.

In order to adequately fill the ventricles, against the increasing pericardial pressure both systemic and pulmonary vascular beds must generate sufficient pressure. In addition cardiac chamber compliance is also reduced by pericardial compression, leading to progressive resistance to filling. So to increase the filling pressure, body starts to conserve fluid, which is a gradual adaptation, hence requires time, which is not possible in acute cardiac tamponade.

As a result in case of gradually developing tamponade, diastolic pressure in both ventricles and pulmonary artery equilibrates with mean right and left atrial pressure at approximately the intra-pericardial pressure. This is the phase at which most patients will have frank exaggeration of respiratory fluctuation in arterial blood pressure (pulsus paradoxus).

Tamponading pericardial fluid compresses the heart throughout systole and diastole. Although the atria fill continuously, blood mainly enters the heart

when blood is leaving it during the right and left ventricular ejection periods, since ventricular ejection expels blood, reducing ventricular volumes. Ejection thus transiently reduces pericardial pressure, transiently increasing transmural pressure. Ejection simultaneously aids atrial filling through enlarging the atria by pulling their "floors" (valve levels) toward the ventricular apices[22].

Like most tamponade induced abnormalities of pressure and flow, transmural pressures are reciprocally reduced and increased during the respiratory phases for the left vs the right heart. Thus, inspiration increases right heart filling at the expense of the left heart with reversal in expiration. In critical tamponade, when cardiac output usually has fallen by at least 30%[21,23.24,25], transmural pressures are, on average, zero (typically between 15 and 30 mmHg within the pericardium and between 15 and 30 mmHg within the heart in euvolemic patients) so that respiratory reciprocation becomes a principal physiologic mechanism contributing at some level to cardiac input and output. A significant component of respiratory reciprocation is the marked shift of the ventricular septum into the left ventricle when inspiration fills the right heart at the expense of the left with reversal on expiration. Clinically, respiratory reciprocation is expressed as pulsus paradoxus.

Echo parameters used to confirm cardiac tamponade:

The criteria[26,27] that suggest cardiac tamponade by echocardiogram are:

1. Right ventricular diastolic and right atrial collapse[9,28,9,30,31].

2. Interventricular septum deviation toward the left ventricular cavity on inspiration.
3. An increase in trans-mitral inflow E wave velocity of more than 25% on expiration and/or increase in tricuspid inflow E wave velocity by greater than 40% on inspiration.
4. Inspiratory variation of peak aortic and pulmonary flow velocities[32].
5. Inferior vena cava diameter and its variation with respiration.

The normal trans-AV valvular flow profile has two peaks - an E and an A wave. The E peak arises due to early diastolic filling. Most filling (70-75%) of the ventricle occurs during this phase. The A peak arises due to atrial contraction, forcing approximately 20-25% of stroke volume into the ventricle. Classically, the E-wave velocity is slightly greater than that of the A wave. As the normal output of ventricles varies with respiration, so varies the peak of Eand A velocity during phases of respiration. This phenomenon is exaggerated in cardiac tamponade and is taken as a marker for tamponade.

Previous studies:

Few animal studies were done to establish the hypothesis that massive pleural effusion can cause cardiac tamponade like physiology, based on the concept of close approximation of both pleura around the heart and the close relationship between intra-pleural and intra-pericardial pressure during normal quite respiration.

Vaska[8] et al demonstrated that right ventricular diastolic collapse[28] was produced by intrapleural instillation of fluid in a canine model. It was observed that the larger the pleural effusion, the higher the pericardial pressure. In the study by Vaska et al, the following figures give relationship between various parameters and intra-pericardial pressure, with instillation of fluid in pericardial space and pleural space.

Figure 4Relationship between pleural and pericardial pressure during intrapleural instillation (dotted lines) and intrapericardial instillation (solid lines) of fluid

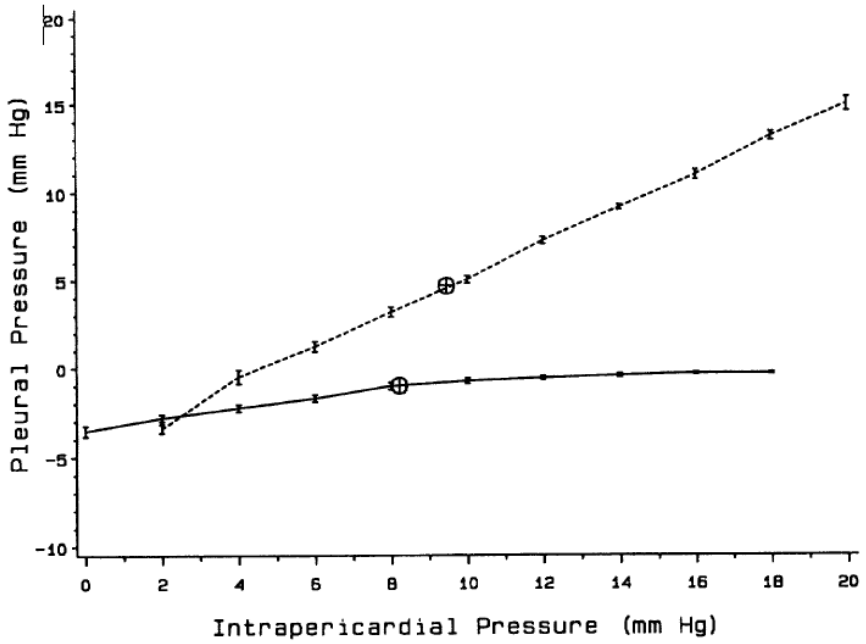
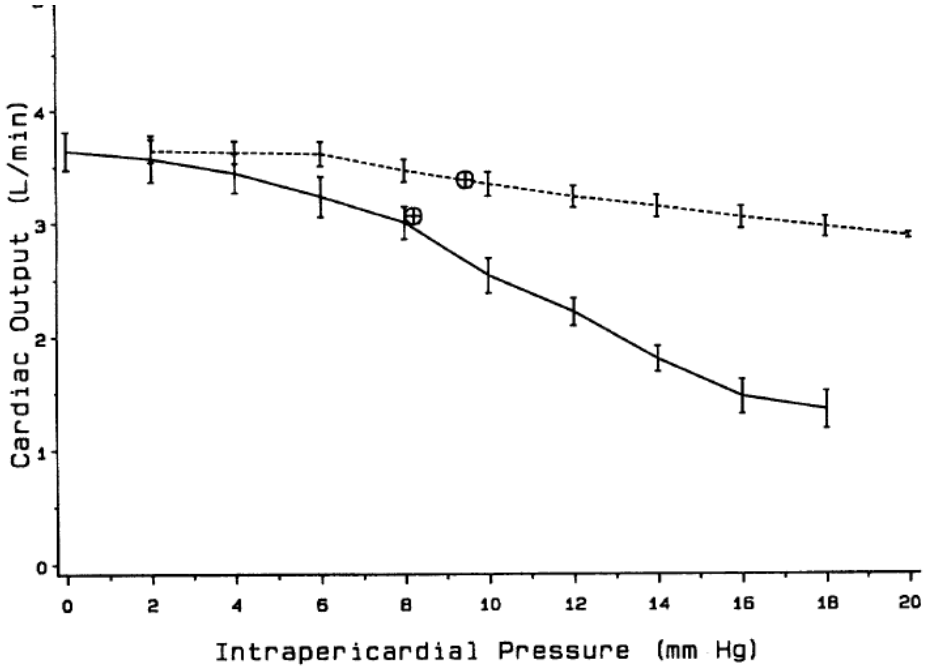


Figure 4 shows that, with intra-pleural instillation of fluid, the relationship between pleural pressure and intra-pericardial pressure rise is linear, whereas with intra-pericardial instillation of fluid, intra-pericardial pressure rises promptly but with minimal increase in pleural pressure.

Figure 5 Showing relationship between intra-pericardial pressure and cardiac output with intra-pleural (dotted lines) and intra-pericardial instillation of fluid (solid lines)



From Figure 5, it is clear that intra-pericardial pressure rises with both intra-pleural and intra-pericardial instillation of fluid, but more steeply with intrapericardial instillation of fluid, compared to intra-pleural instillation. When cardiac output is compared, intra-pleural fluid instillation is better tolerated than intra-pericardial instillation of fluid, due to less abrupt fall in cardiac output in the former.

Wrisleyetal[28] had demonstrated marked diastolic collapse of right atrium without hemodynamic compromise in dogs. Similar results were obtained from animal studies conducted by Kaplan et al[29] and Venkatesh et al[33].

Echocardiographic evidence of cardiac tamponade in patients with pleural effusion without pericardial effusion was shown by Arasadaniantz et al[9], who

reported that in 116 patients with pleural effusion with no pericardial effusion 18% had evidence of cardiac chamber collapse on echocardiography.

Traylor et al[34] evaluated 37 patients with large pleural effusion with echocardiographic and clinical signs of cardiac tamponade[9]. In their study, the patients were divided into two groups. Group 1: Pleural effusions occupying less than a hemithorax, (n = 10). Group 2: Effusions greater than a hemithorax on chest X ray, (n = 27). Group 1 patients had no clinical or echocardiographic signs of cardiac tamponade. However, in Group 2, 8 subjects had elevated JVP, 8 had pulsus paradoxus, 6 had RV diastolic collapse, and 23 had flow velocity paradoxus. Those signs of cardiac tamponade resolved completely in 20 patients after thoracentesis. Pericardiocentesis was not performed in any of these patients. In terms of sidedness of pleural effusion, group 2 showed that 11 patients had left, 8 had right, and 8 had bilateral pleural effusions. Although the study clearly demonstrated size of pleural effusion is an important factor, location of pleural fluid did not have a significant impact on cardiac tamponade physiology.

In the study by Traylor et al. in group 2, 8 patients had small pericardial effusion (not quantitated). Studies had shown that size of pericardial effusion is not linearly related to intra-pericardial pressure and any acute increase in quantity of pericardial fluid can result in cardiac tamponade, rather than absolute quantity of fluid increase. So as there are no clues to rapidity of fluid accumulation in study by Traylor et al. there exists possibility of pericardial

effusion being a confounder. So this study was planned to exclude all pericardial effusions, however trivial they may be.

Other assumptions and procedures were planned similar Traylor et.al[34].

Right ventricular diastolic collapse is a sensitive and specific indicator for the early detection of hemodynamically important pericardial effusions and is used in experimental studies on pleural effusion. Traylor et al had noted right atrial collapse, which is the commonly used marker for tamponade physiology, as RVDC occurs early in the course of tamponade than right atrial collapse.

In addition to flow velocity paradoxus across AV valves all possible parameters involved in cardiac tamponade like pulmonary artery and aortic respiratory variations RA,RV,LA sizes before and after thoracentesis were included in this study.

Further improvement, in defining the concept can be done by future studies with invasive monitoring of pleural pressure, pericardial pressure and cardiac chambers before and after thoracentesis and correlating them.

MATERIALS AND METHODS

Setting : In-patient department,
Institute of Internal Medicine,
Rajiv Gandhi Government General Hospital,
Madras Medical College, Chennai.
Echocardiography lab,
Department of cardiology,
Madras Medical College, Chennai.

Sampling technique	:	Convenient sampling
Ethical committee approval	:	Obtained
Design of the study	:	Prospective study
Period of study	:	April 2011 to November 2011
Sample size	:	40 patients

Inclusion Criteria:

1. Age > 12
2. Unilateral or bilateral massive pleural effusion of non-cardiac etiology

Exclusion Criteria:

1. Pleural effusion secondary to cardiac disease
2. Constrictive pericarditis
3. Pericardial effusion

Case Definition:

In our study, the criteria used for classifying massive pleural effusion is any pleural effusion with chest x-ray postero-anterior view showing homogenous opacity greater than or equal to ¾ of one or both hemithorax.

METHODOLOGY AND INVESTIGATION DETAILS OF PATIENTS

After obtaining informed and written consent, patients above the age of 12 years who had unilateral or bilateral massive pleural effusion of non-cardiac etiology were included in the study. Patients with pleural effusion secondary to cardiac disease and patients with constrictive pericarditis, patients with mild and moderate pleural effusion (less than ¾ of a hemithorax) were excluded from the study. Patients were selected from inpatients wards in Institute of internal medicine, where they were evaluated for pleural effusion.

Relevant history, physical examination, chest X ray, electrocardiogram were done for all the patients. Patients with JVP above 3 cm from sternal angle are noted.Spo2, pulse rate, respiratory rate, systolic and diastolic blood pressure were taken and patients with pulsus paradoxus are noted. All the patients underwent detailed echocardiographic study. The following echocardiographic parameters were done which includesflow velocity across tricuspid valve, mitral valve, pulmonary valve, and aorta, size of all the chambers and pulmonary artery pressures were measured.

All the patients underwent therapeutic thorocentesis for massive pleural effusion.A repeat chest x ray was taken to assure that the pleural effusion remained less than a ½ of a hemithorax and all the clinical and echo parameters were measured again within 24 hours. Pre and post therapeutic echo parameters were compared.

Data Analysis Methods:

Statistical analysis was done withGraphpadinstat3. Values obtained before and after intervention were analysed using paired t-test and significance of sidedness of pleural effusion, for development of cardiac tamponade physiology was analysed using Fisher's exact test.

RESULTS

Patients' demographics and clinical factors:

Forty patients who had massive unilateral or bilateral pleural effusion were included in our study. Out of 40 patients, 32were male and 8 were female patients. 30patients had right sided, 7 had left sided and 3 had bilateral pleural effusion. 25 patients had pleural effusion secondary to tuberculosis and 14 secondary to malignancy 1 due to decompensated liver disease with moderate ascites. Mean age of the patients was 46.6.

Figure 6 Age distribution

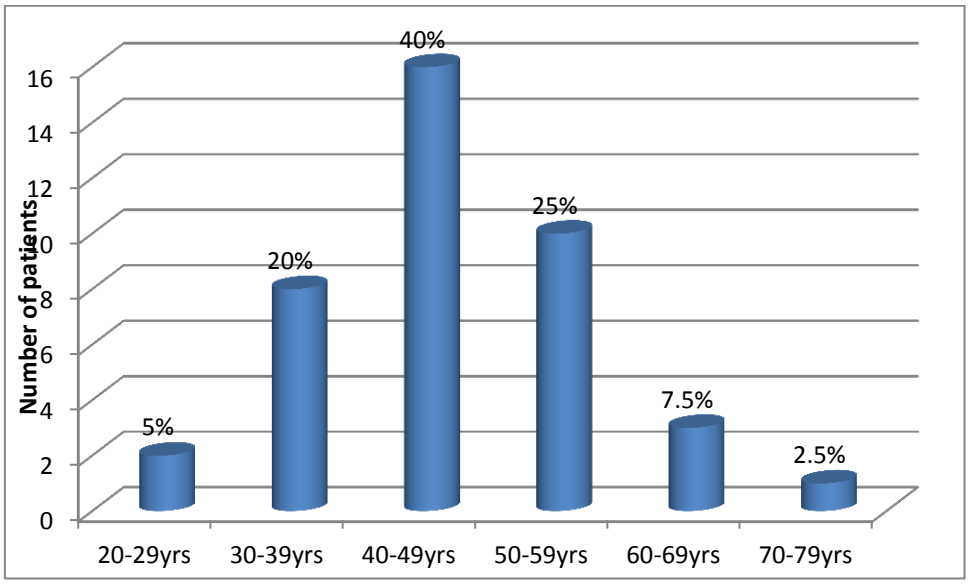


Figure 7 Sex distribution

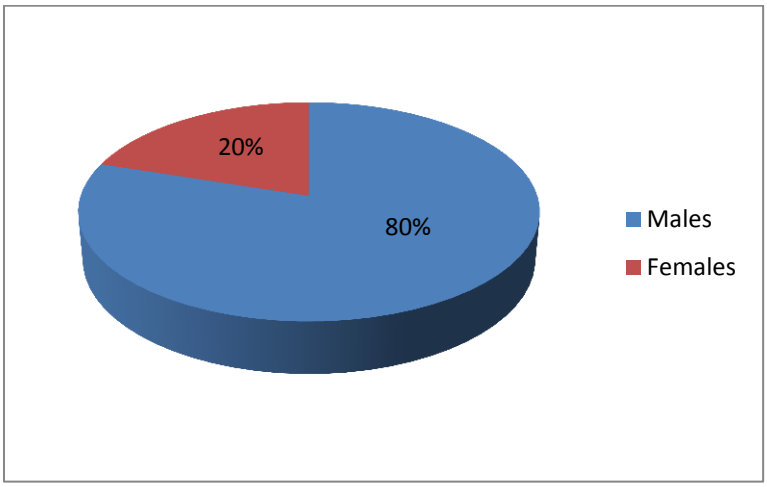


Figure 8 Sidedness of pleural effusion

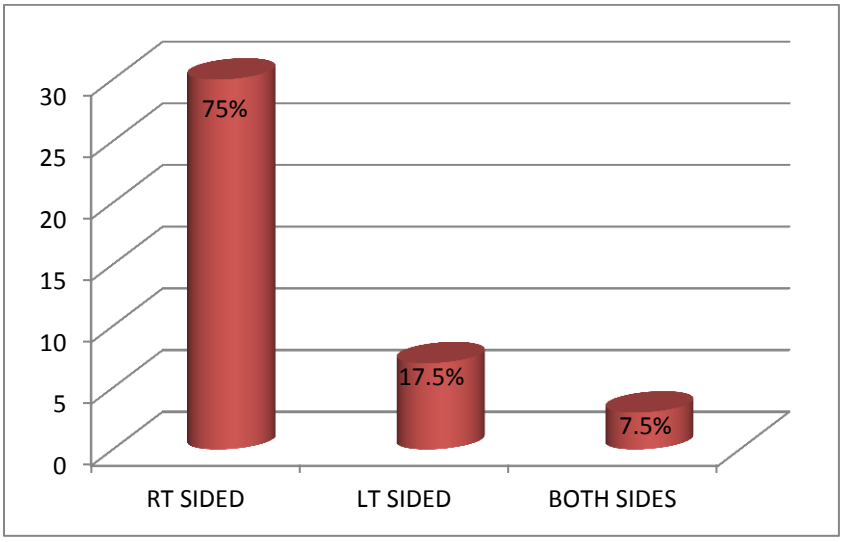


Figure 9 Etiologies of pleural effusion

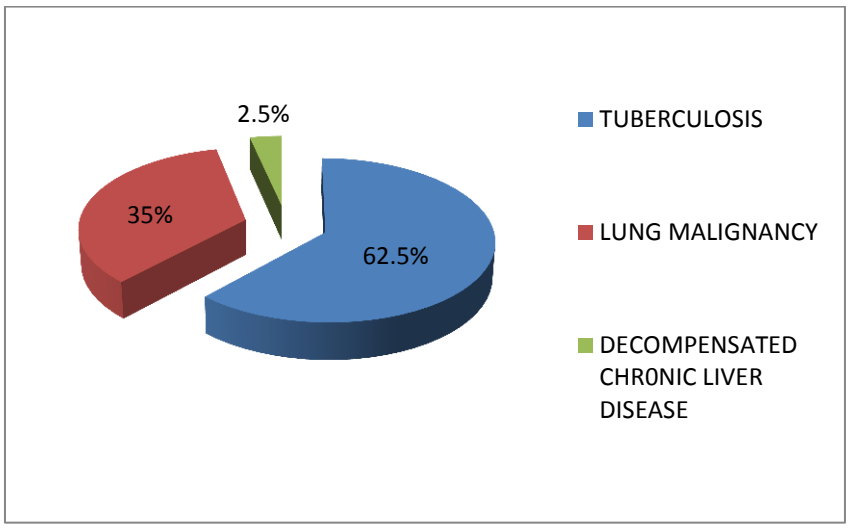


Figure 10 X-ray of a patient with right sided massive pleural effusion

Out of 7 patients with left sided massive pleural effusion, 4 had elevated JVP and all the 4 patients also demonstrated pulsus paradoxus. In case of right sided pleural effusion, out of 30 cases with massive pleural effusion 13had

elevated JVP and 18demonstrated pulsus paradoxus, whereas all three patients who had bilateral pleural effusion with at least one hemithorax meeting the inclusion criteria had both elevated JVP and pulsus paradoxus.

Figure 11 Elevated JVP in the study group

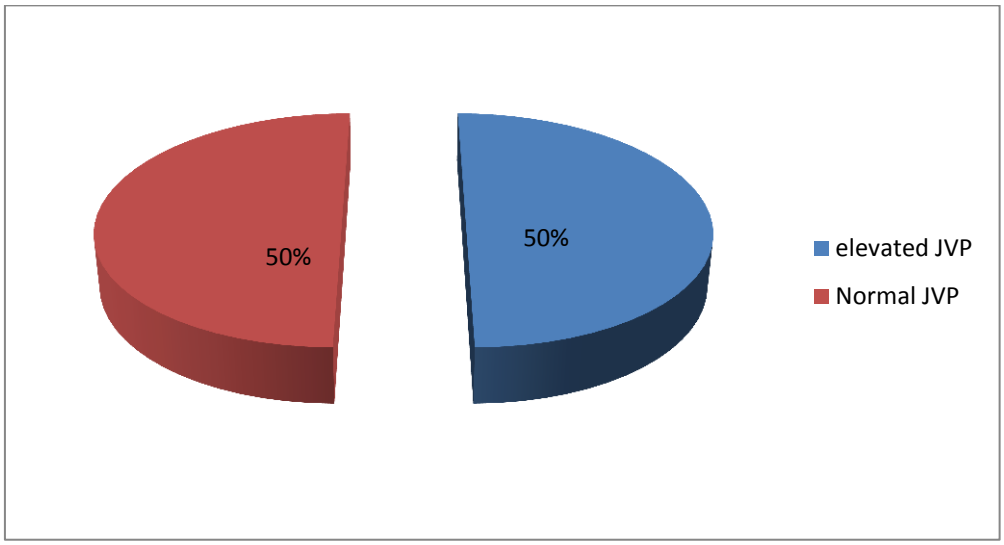


Figure 12 Pulsus paradoxus in the study group

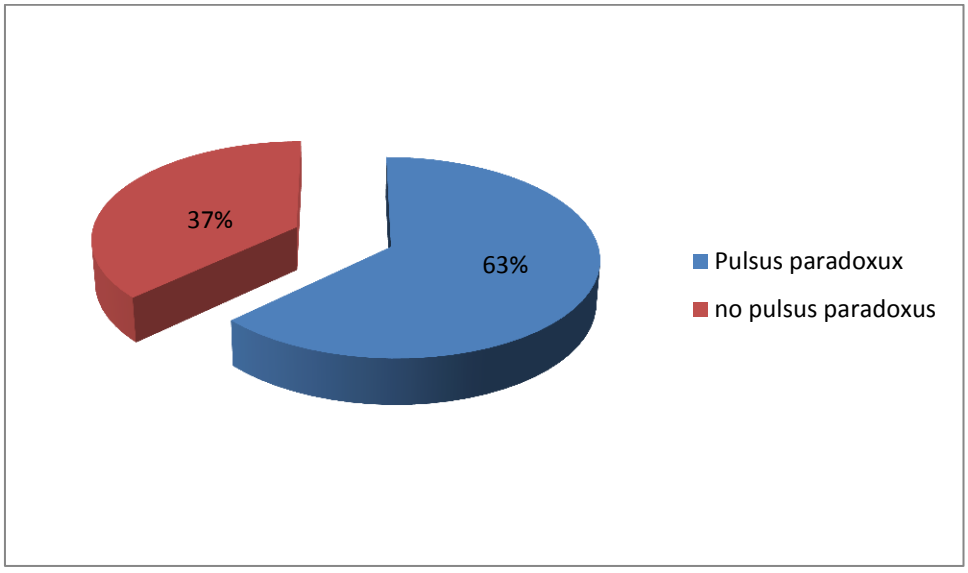
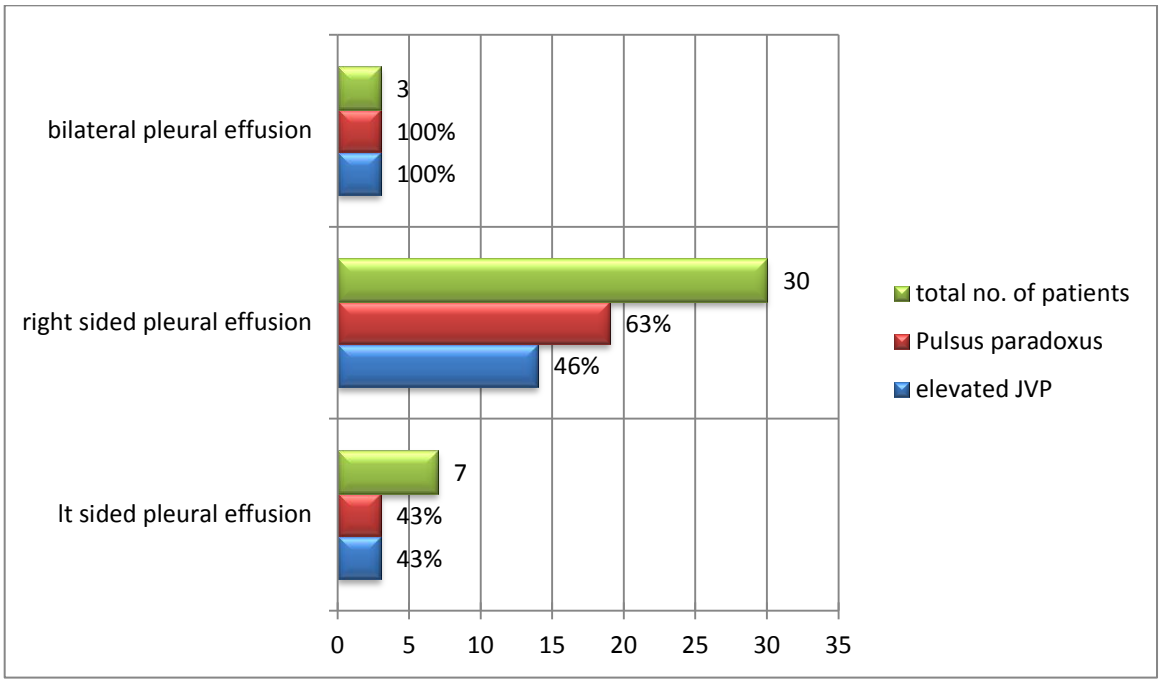


Figure 13 Relative percentage of patients in each group with pulsus paradoxus and elevated JVP



As shown in table 1, before thorocentesis, SpO_2 (mean ± SD) was97%±1.3%, pulse rate was101.75±4.8, systolic blood pressure was110.2±9.2, and diastolic blood pressure was 75±7.3. After thorocentesis, $SpO_2$was 98%±0.9, pulse rate was 93.43±3.5, systolic blood pressure was114.40±7.6, and diastolic blood pressure was 78.60±4.6.

Table 1

Parameter	Before Thoracentesis	After Thoracentesis
SPO_2	97%±1.3%	98%±0.9%
Systolic BP	110.2±9.2	114.40±7.6
Diastolic BP	75±7.3	78.60±4.6
Pulse rate	101.75±4.8	93.43±3.5

Figure 14 Mean SpO2, systolic BP, diastolic BP, pulse rate before and after thoracentesis

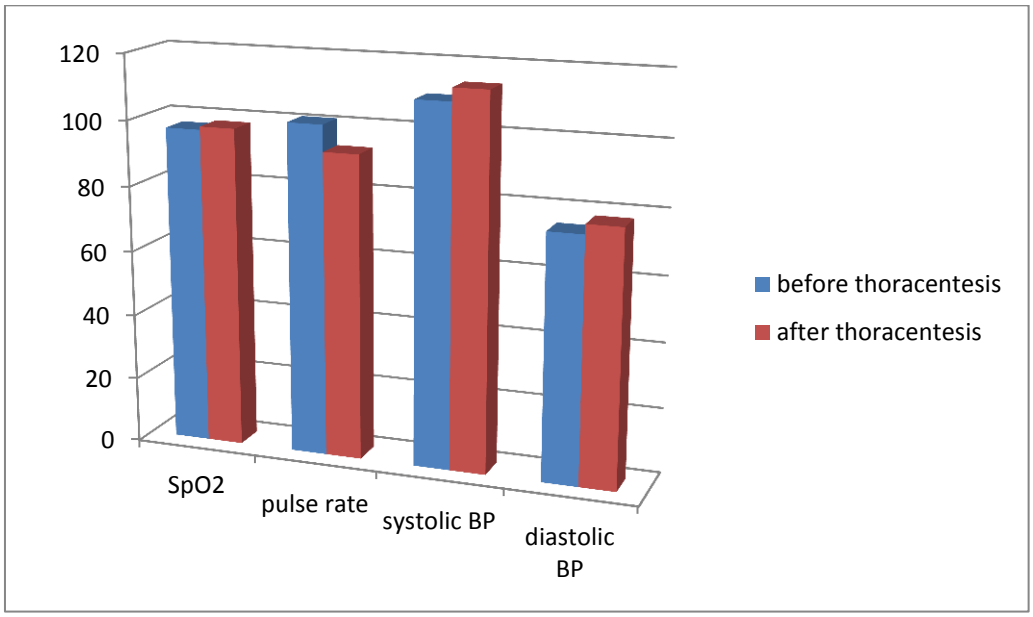


Figure 15 SpO2 values before and after thoracentesis

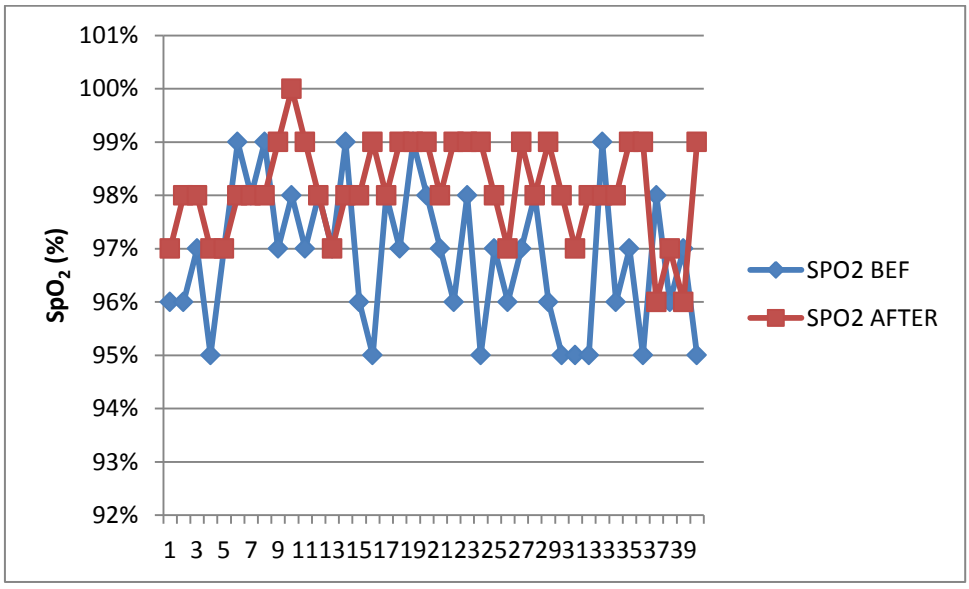


Figure 16 Pulse rate in study patients before and after thoracentesis

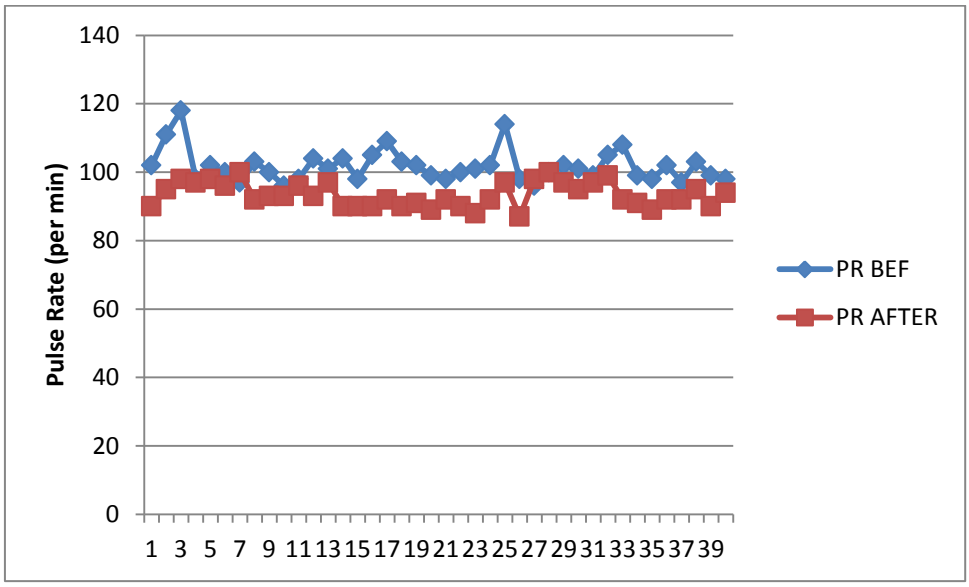


Figure 17 Systolic blood pressure in study patients before and after thoracentesis

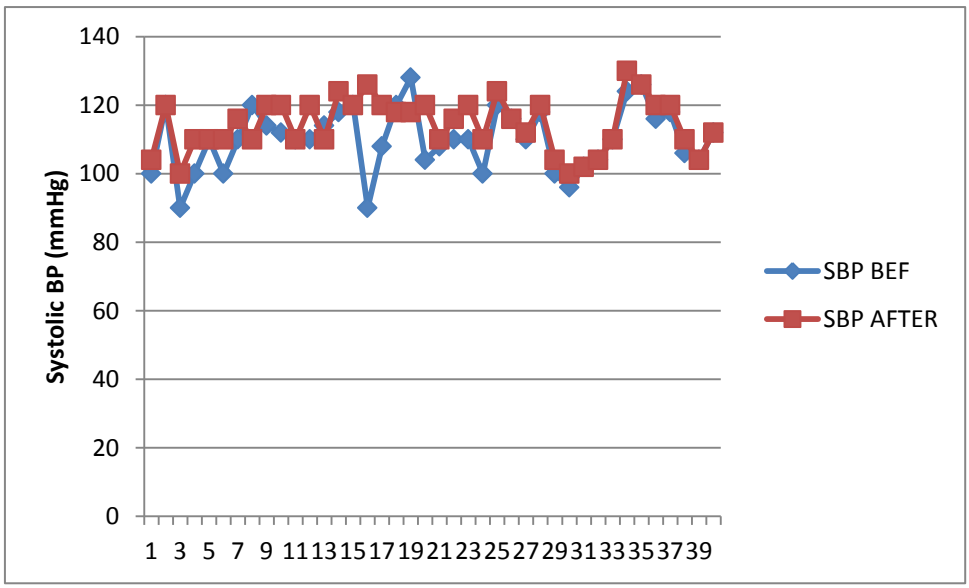
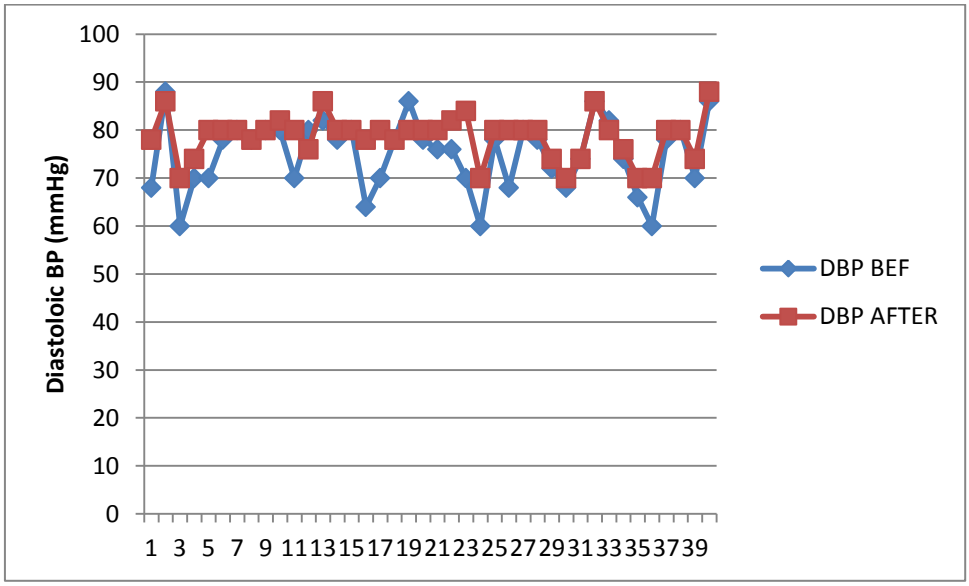


Figure 18 Diastolic blood pressure in study patients before and after thoracentesis



Echocardiographic parameters:

Chamber size before thoracentesis, mean right atrial size 3.40cm, mean left atrial size 3.5cm, mean left ventricular systolic diameter 3.12 cm, mean left ventricular diastolic diameter 5.1cm. Mean right ventricular measurements at the basal, mid and base to apex were 2.4 cm, 2.93 cm, and 7.37 cm respectively. After thoracentesis, mean right atrial size 3.32 cm , mean left atrial size 3.42 cm, mean left ventricular systolic diameter 3.26cm, mean diastolic diameter 5.2 cm, mean right ventricular measurements at the basal, mid and base to apex measurements 2.45 cm, 2.98 cm,7.45 cm respectively and 6 patients had right atrial collapse.

Figure 19 Echo showing right atrial collapse[35]

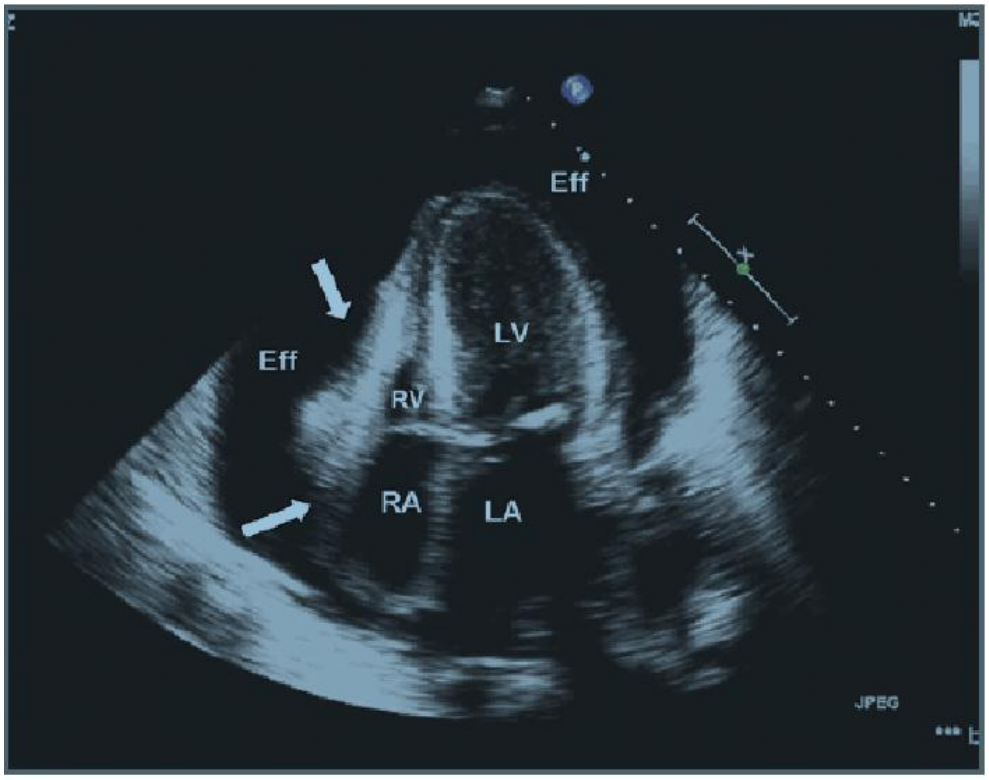


Figure 20 right atrial size before and after thoracentesis

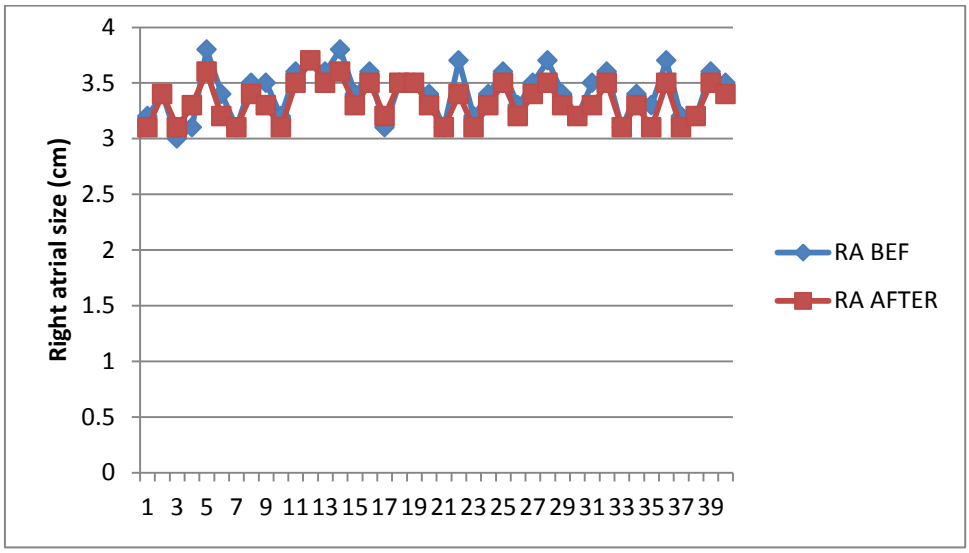


Figure 21 left atrial size before and after thoracentesis

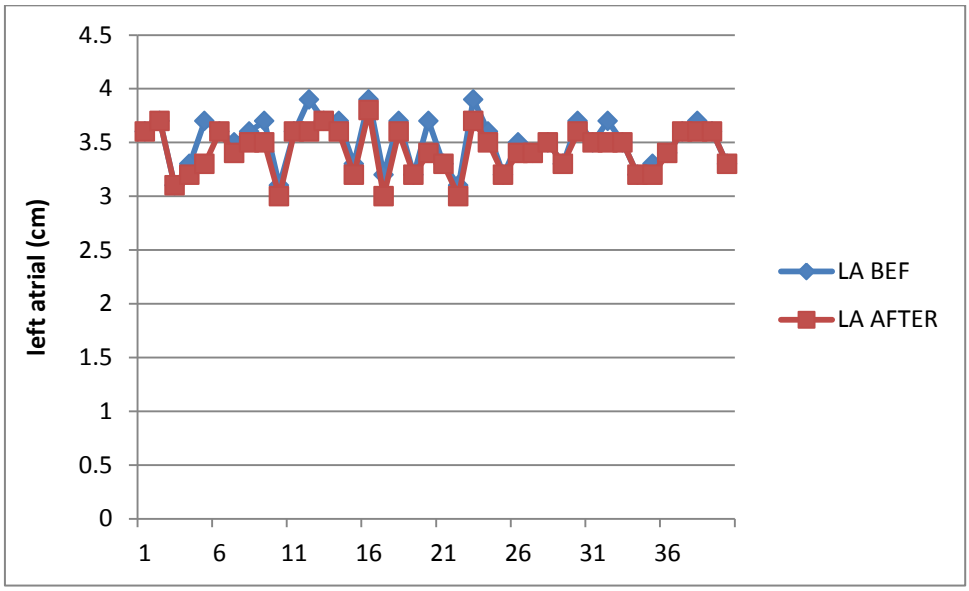


Figure 22 left ventricular dimension before and after thoracentesis

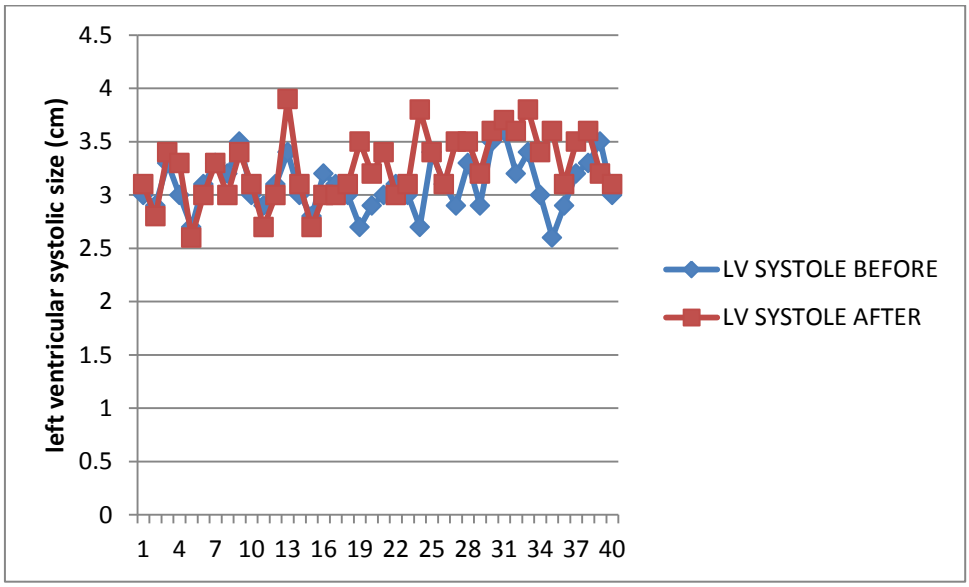


Figure 23 left ventricular diastolic size before and after thoracentesis

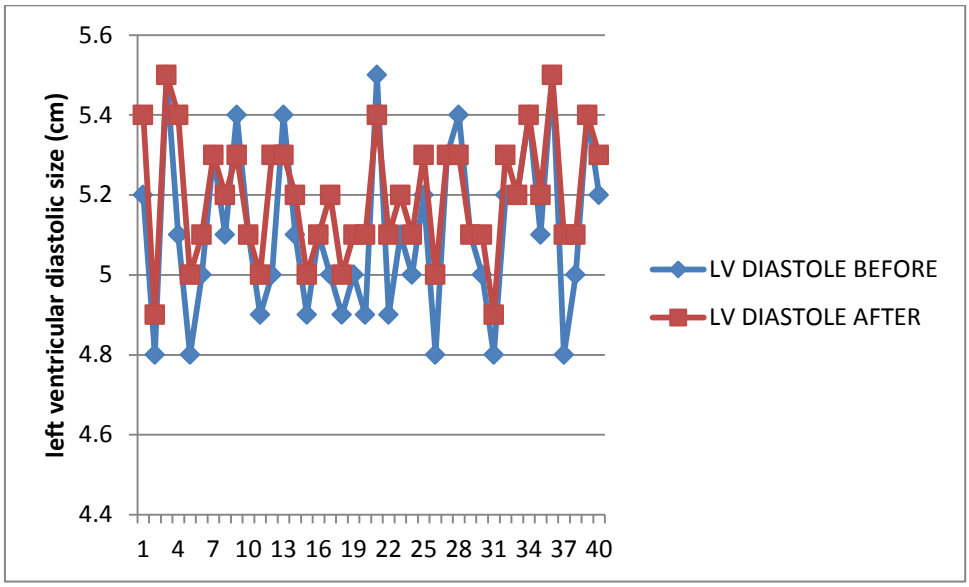


Figure 24 right ventricular size at base before and after thoracentesis

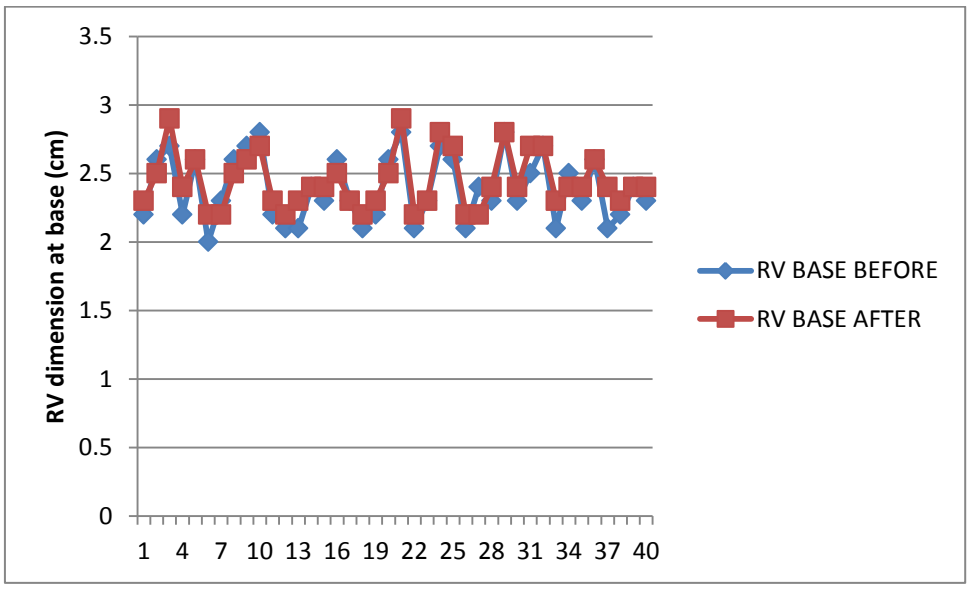


Figure 25 right ventricular size at mid-level before and after thoracentesis

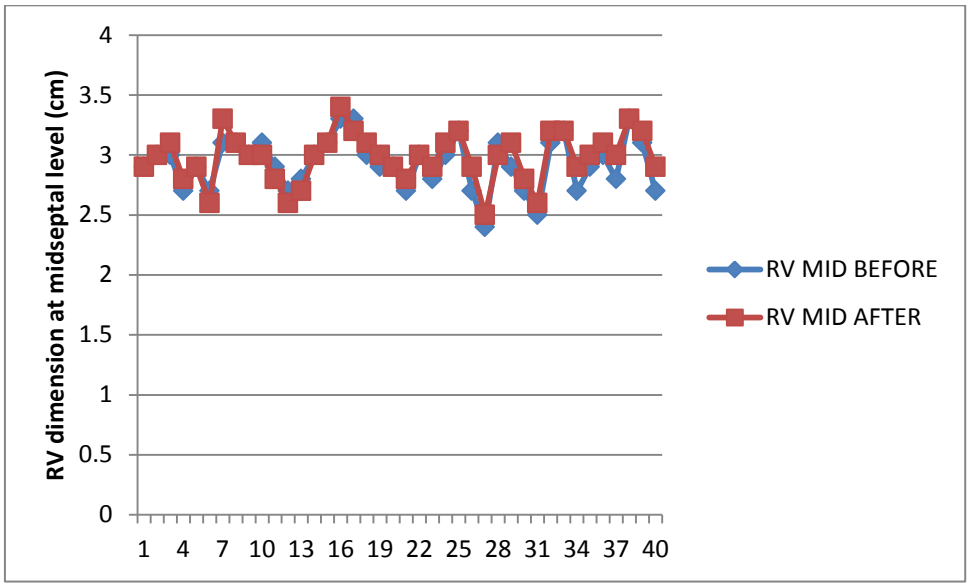


Figure 26 right ventricular size (base to apex) before and after thoracentesis

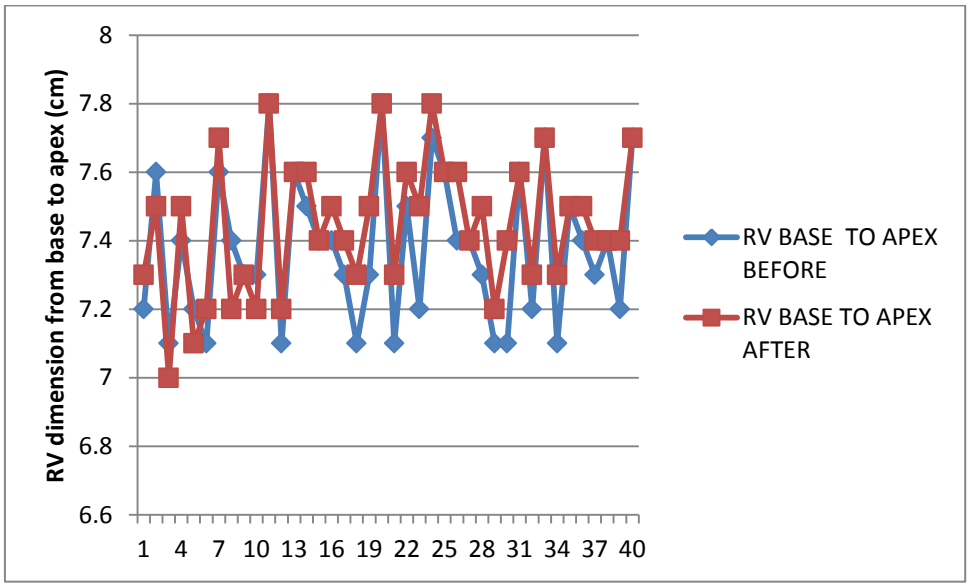


Table 2 showing mean chamber size before and after thoracentesis

Parameter	Before Thoracentesis (cm)	After Thoracentesis (cm)
mean RA size	3.40	3.32
mean RV size – basal	2.4	2.45
mean RV size - mid	2.93	2.98
mean RV size – base to apex	7.37	7.45
mean LA size	3.5	3.42
mean LV systolic diameter	3.12	3.26
LV diastolic diameter	5.1	5.2

Before thoracentesis mean flow velocity across tricuspid valve during inspiration (E) - 74.47cm/s,(A) - 63.77cm/s; during expiration (E) - 52 cm/s, (A) - 42.825 cm/s. Mean flow velocity across mitral valve during expiration (E) - 70.7 cm/s, (A) - 72.4 cm/s; during inspiration (E) - 53.6 cm/s , (A) - 57.8 cm/s. Mean flow velocity across pulmonary artery during inspiration was 85.65 cm/s, and during expiration was 63.27cm/s. Mean flow velocity across aorta during expiration was 86.2 cm/s and during inspiration was 71.68 cm/s.

After thoracentesis, mean flow velocity across tricuspid valve during inspiration (E) - 68.4 cm/s, (A) - 62 cm/s; during expiration (E) - 52.2 cm/s, (A) - 47.5 cm/s. Mean flow velocity across mitral valve during expiration (E) - 65.3

cm/s, (A) - 70.3 cm/s; during inspiration (E) - 54.55 cm/s, (A) - 58.32 cm/s. Mean flow velocity across pulmonary artery during inspiration was 81.46 cm/s, and during expiration was 70 cm/s. Mean flow velocity across aorta during expiration was 79.5cm/s and during inspiration was 70 cm/s.

Figure 27 measurement of mitral E, A velocities

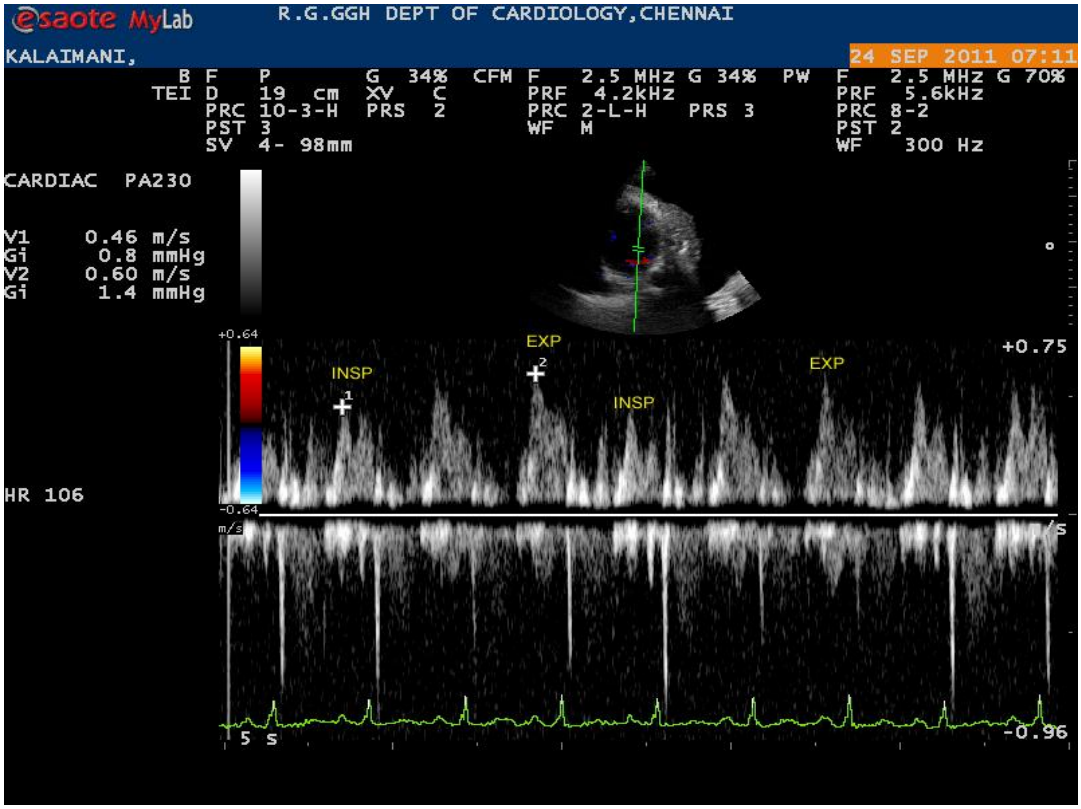


Figure 28 measurement of tricuspid E, A velocities

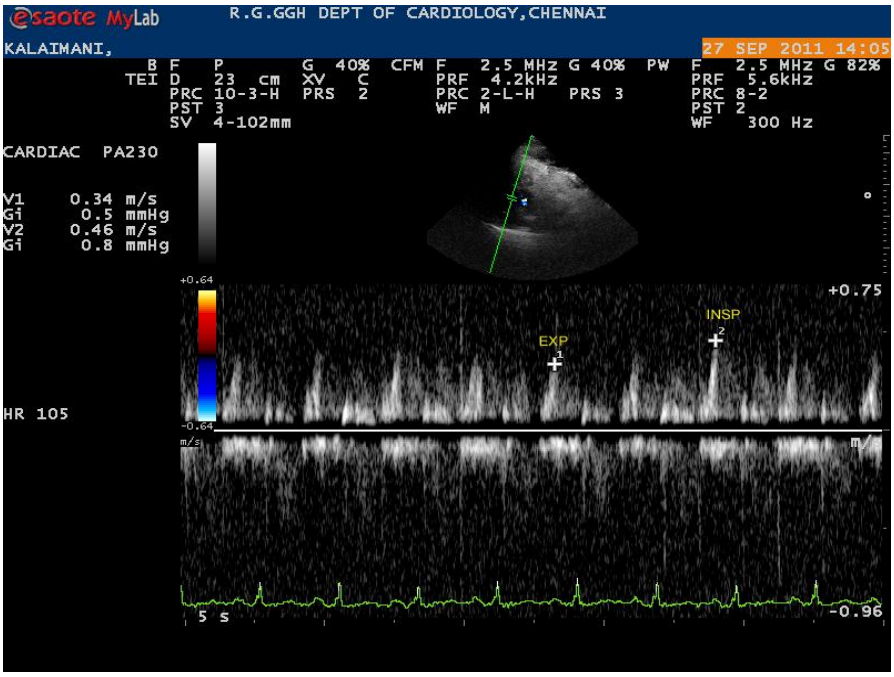


Figure 29 measurement of aortic flow velocities

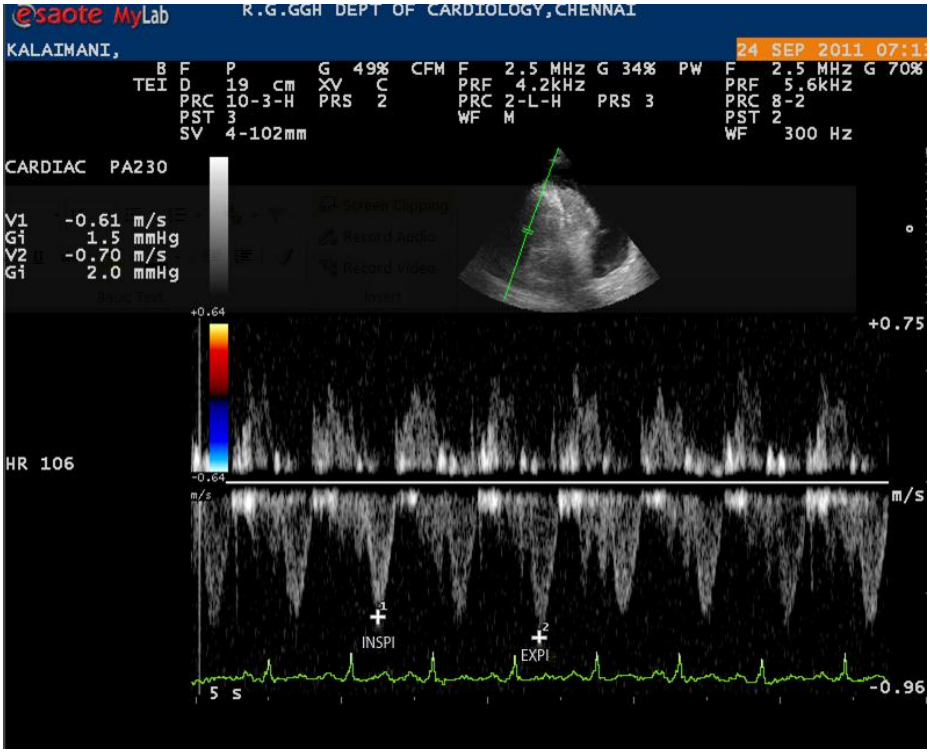


Figure 30 measurement of pulmonary artery flow velocities

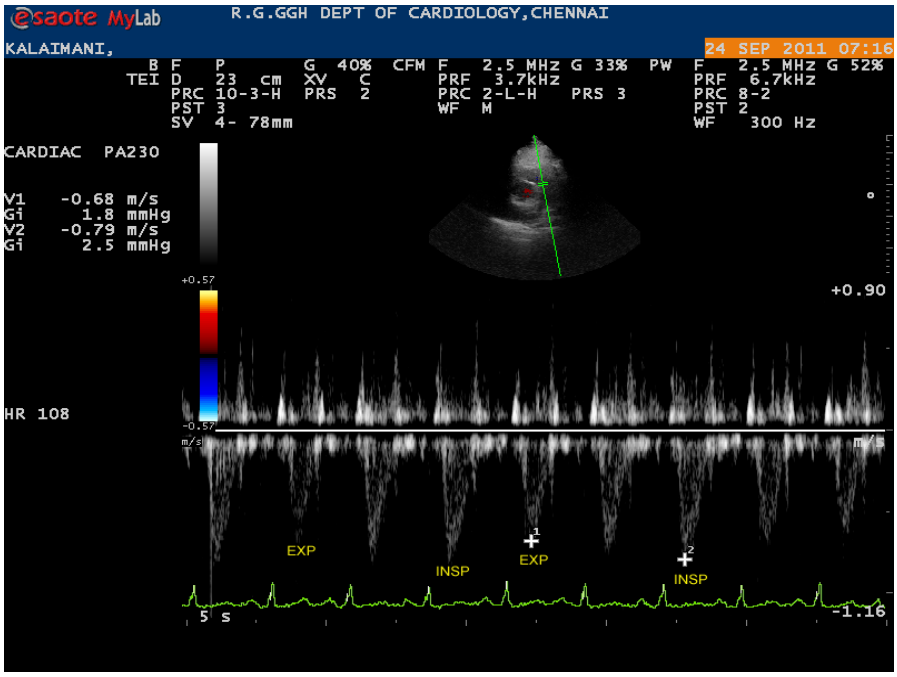


Figure 31 measurement of LV function

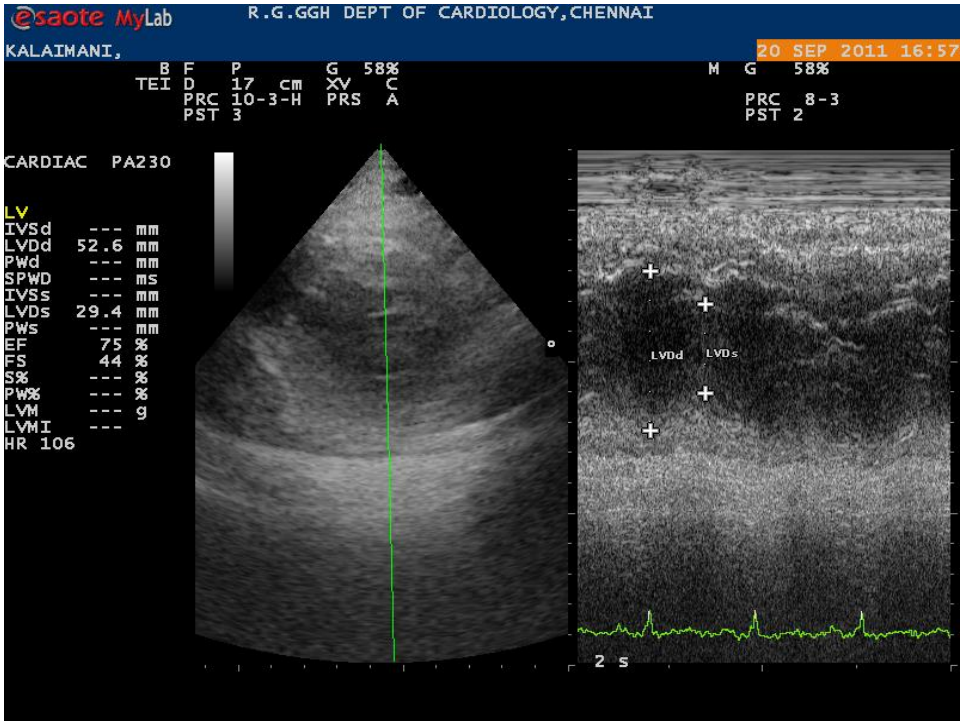


Figure 32 mitral E velocity variation before and after thoracentesis

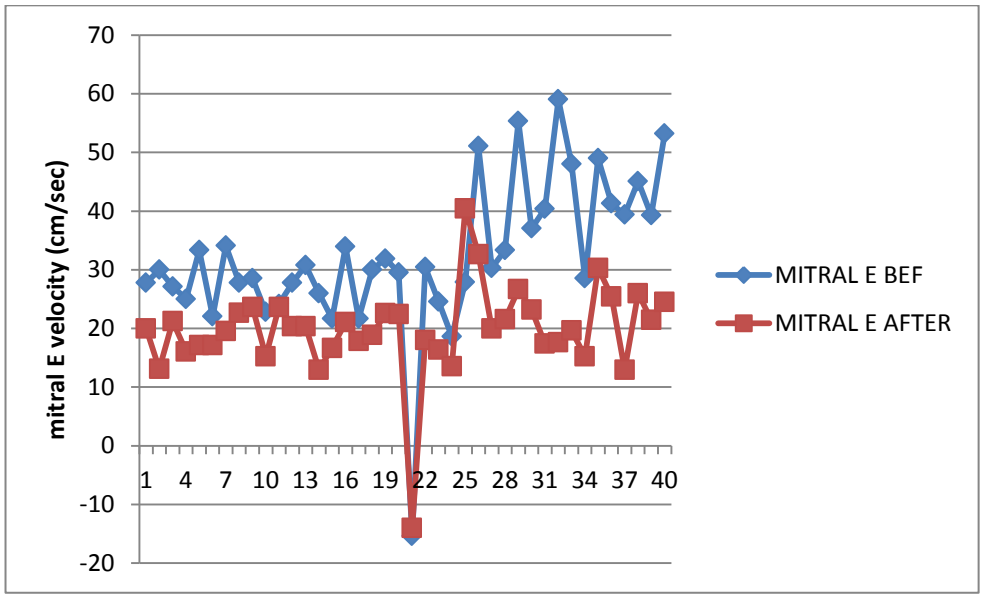


Figure 33 mitral A velocity variation before and after thoracentesis

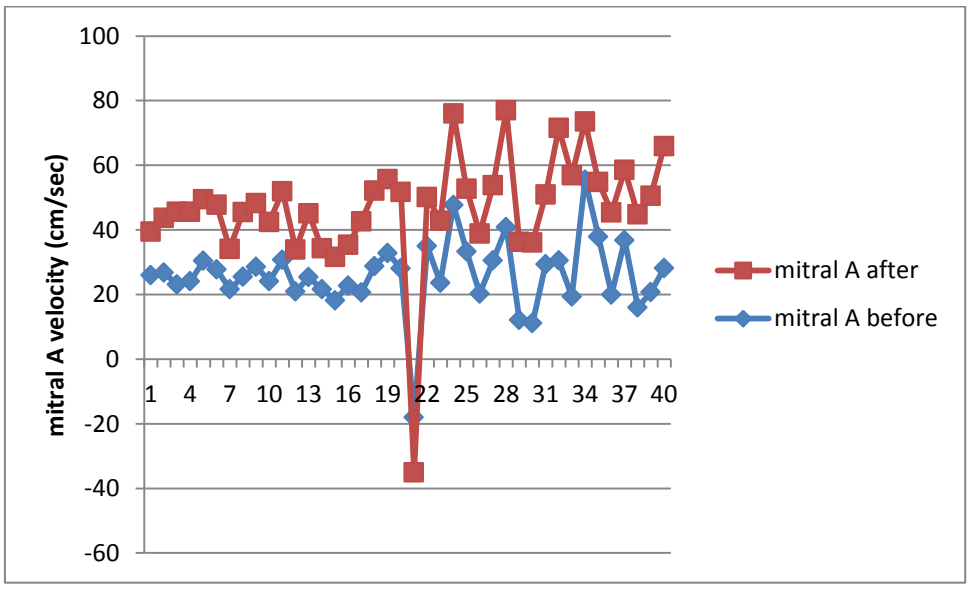


Figure 34 tricuspid E velocity variation before and after thoracentesis

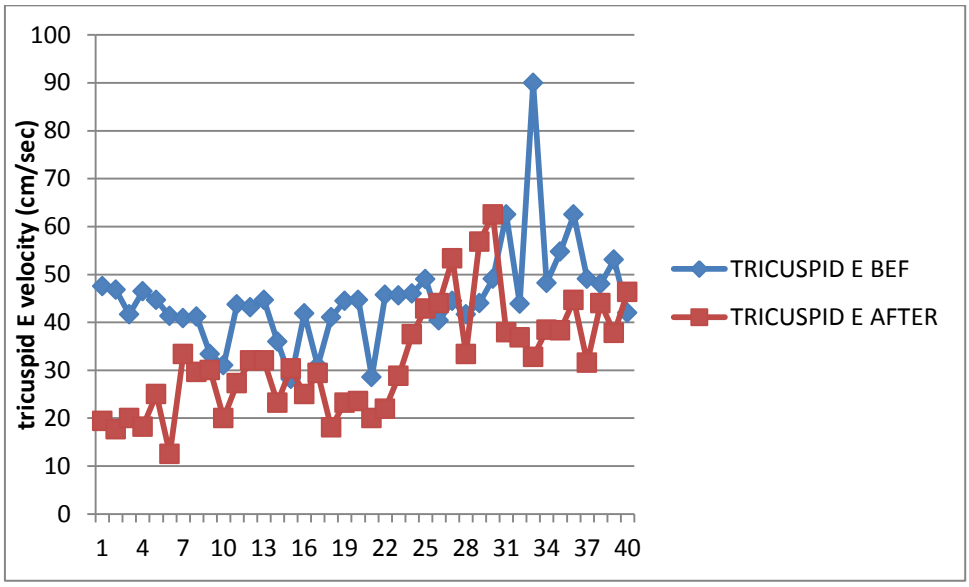


Figure 35 tricuspid A velocity variation before and after thoracentesis

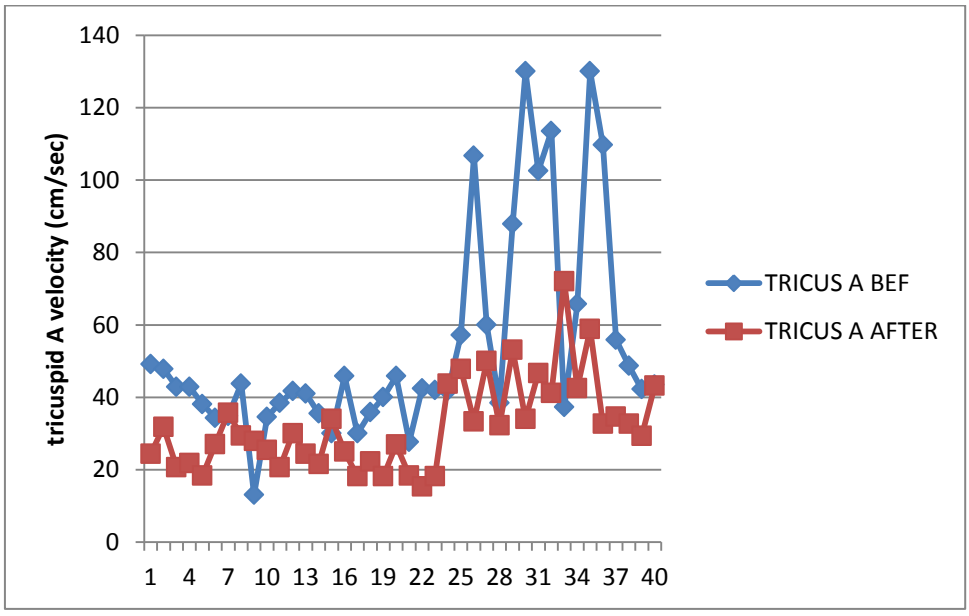


Figure 36 pulmorary artery flow velocity variation before and after thoracentesis

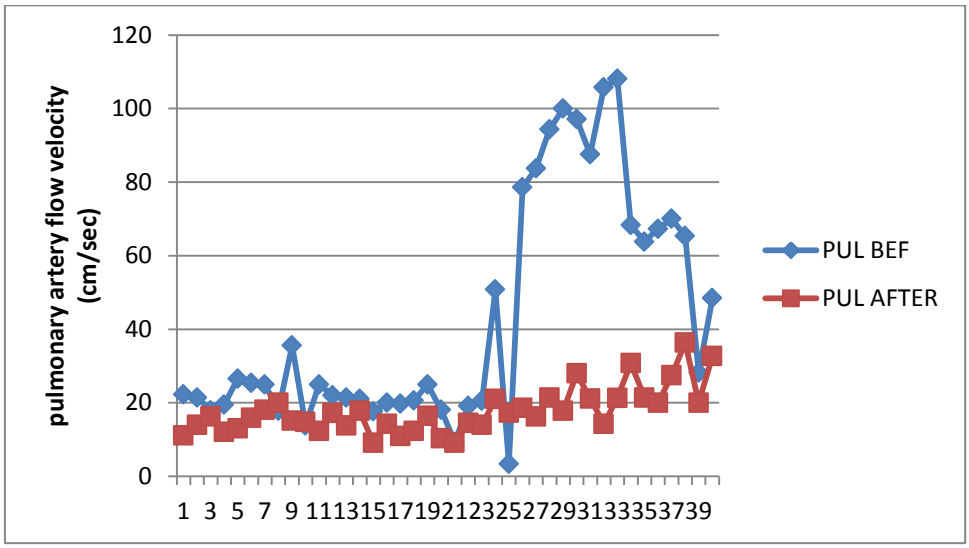


Figure 37 aortic flow velocity variation before and after thoracentesis

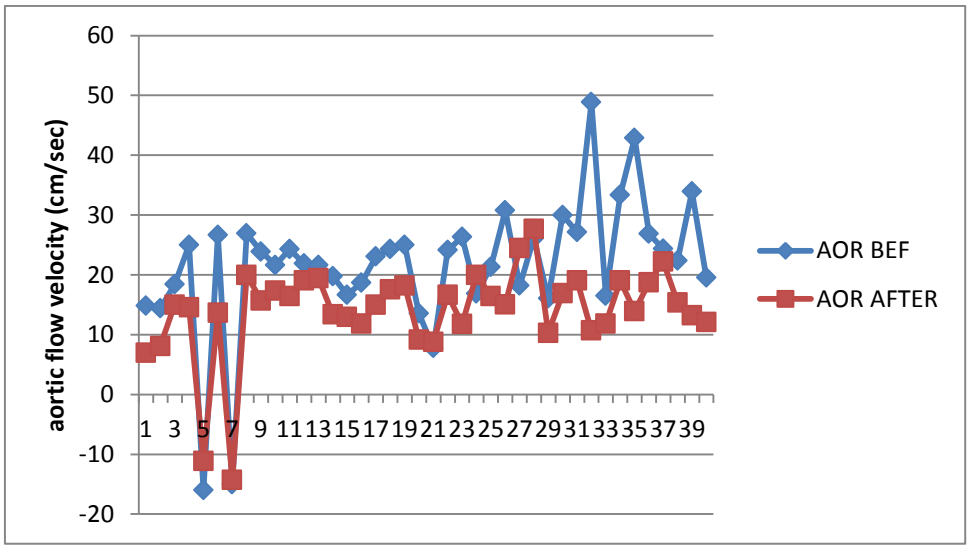


Figure 38 mean respiratory variation across AV valves, pulmonary and aortic valve

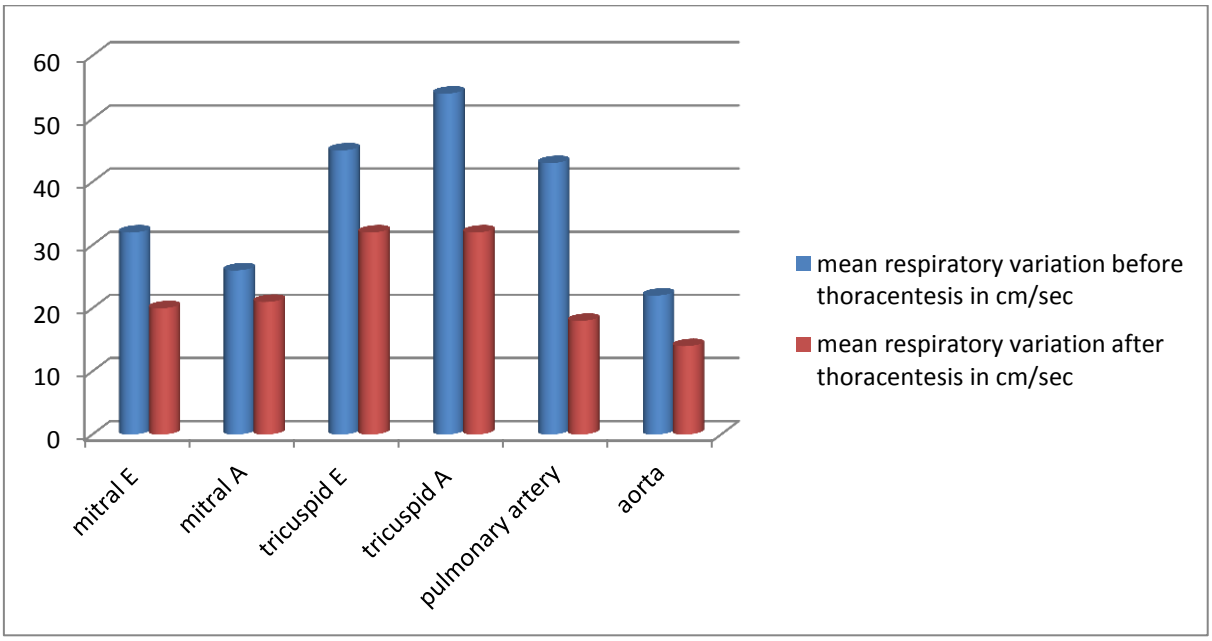


Table 3 showing mean flow velocities across A-V valves and semilunar valves before and after thoracentesis

Table 3

Parameter (Mean Respiratory Variation)	BeforeThoracentesis	Normal Values	After Thoracentesis
Tricuspid E	45.04±10.4%	<40%	32±11.3%
Tricuspid A	53.71±28%	<40%	32.08±12.5%
Mitral E	32.30±12 %,	<25%	19.78±7.8%
Mitral A	26±11.2%	<25%	21±9.3%
Pulmonary artery	42.63±31.3%	<10%	17.70±6.2%
Aorta	21.57±11.4%	<15%	14.08±7.6%

Mean flow velocity respiratory variations, across tricuspid valve before thoracentesis and after thoracentesis were E- 45.04±10.3, E - 32±11.3% (p value < 0.001); A - 53.71±29%, A- 32.08±12.5% (p < 0.001); across mitral valve E - 32.30±12.7 %, E - 19.78±7.8% (p < 0.001); A - 26±11.2 %, A- 21±9.3% (p < 0.001); across pulmonary artery 42.63±31.3%, and 17.70±6.2% (p < 0.001); across aorta 21.57±11.4% , 14.08±7.6 % (p < 0.001). The cut off values to designate the respiratory variations in flow velocities as remarkable and representative of cardiac tamponade, for mitral, tricuspid, pulmonary and aorta were >25%, >40%, >10%, >15% respectively.

Table 4

		Mean	**N**	**Standard Deviation**
Pair 1	Mitral E BTC	0.3230	40	0.127
	Mitral E ATC	0.1978	40	0.077
Pair 2	Mitral A BTC	0.2597	40	0.1122
	Mitral A ATC	0.2099	40	0.093
Pair 3	Tricuspid E BTC	0.4504	40	0.1035
	Tricuspid E ATC	0.3199	40	0.1135
Pair 4	Tricuspid A BTC	0.5371	40	0.29
	Tricuspid A ATC	0.3209	40	0.13
Pair 5	Pulmonary BTC	0.4263	40	0.31
	Pulmonary ATC	0.1771	40	0.062
Pair 6	Aorta BTC	0.2157	40	0.11
	Aorta ATC	0.141	40	0.075

BTC- before thoracentesis

ATC - after thoracentesis

Table 5

		N	Std. Error Mean	Two tailed 'P' value
Pair 1	Mitral E BTC	40	0.02	0.000
	Mitral E ATC	40	0.01	
Pair 2	Mitral A BTC	40	0.018	0.006
	Mitral A ATC	40	0.014	
Pair 3	Tricuspid E BTC	40	0.016	0.000
	Tricuspid E ATC	40	0.018	
Pair 4	Tricuspid A BTC	40	0.046	0.000
	Tricuspid A ATC	40	0.02	
Pair 5	Pulmonary BTC	40	0.050	0.000
	Pulmonary ATC	40	0.01	
Pair 6	Aorta BTC	40	0.02	0.000
	Aorta ATC	40	0.012	

From table 4, it is clear that the average respiratory variation across AV valves, pulmonary valve, and aortic valve, before thoracentesis showed values found in cardiac tamponade and after thoracentesis the values reduced to normal range, which was statistically significant, implying that massive pleural effusion can cause cardiac tamponade physiology

Table 6

		Mean	N	Std. Deviation	Std. Error Mean	Two tailed 'P' value
Pair 1	SPO_2 BTC	97.2609	40	1.3	0.25318	0.348
	SPO_2 ATC	98.2609	40	0.9	0.16890	
Pair 2	PR BTC	101	40	4.8	0.75	0.000
	PR ATC	93	40	3.52	0.56	
Pair 3	SYS BP BTC	110	40	9.2	1.45	0.001
	SYS BP ATC	114.4	40	7.64	1.20	
Pair 4	DIA BP BTC	75	40	7.3	1.15	0.000
	DIA BP ATC	78.6	40	4.56	0.72	

BTC – before thoracentesis

ATC- After thoracentesis

From the table 5, it is clear that for Spo2, pulse rate, systolic and diastolic blood pressure before and after thoracentesis, on correlation by statistical analysis got p values as 0.348, 0.0001, 0.001, and 0.0001 respectively.

Regarding chamber size changes before and after thoracentesis, the right atrium, left atrium, left ventricle systolic and diastolic, right ventricle basal, mid, base to apex, p values are 0.001, 0.0001,0.9, 0.0001,0.0056,0.0021,0.0003 respectively, which is depicted in tables 6 and 7.

Using Fisher's exact test for statistical significance, there is no significance for sidedness of pleural effusion on cardiac tamponade physiology in our study and the sample population is also small, to represent a true validation.

Table 7

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	RA cm BTC	3.41	40	0.2179	0.034
	RA cm ATC	3.33	40	0.1728	0.027
Pair 2	LA cm BTC	3.50	40	0.23	0.04
	LA cm ATC	3.42	40	0.21	0.03
Pair 3	LV sys BTC	3.1	40	0.245	0.04
	LY sys ATC	3.26	40	0.315	0.05
Pair 4	LV dias BTC	5.11	40	0.21	0.03
	LV dias ATC	5.19	40	0.16	0.02
Pair 5	RV basal BTC	2.4	40	0.24	0.04
	RV basal ATC	2.45	40	0.20	0.03
Pair 6	RV mid BTC	2.93	40	0.21	0.033
	RV mid ATC	2.98	40	0.205	0.032
Pair 7	RV base-apex BTC	7.375	40	0.213	0.033
	RV base-apex ATC	7.445	40	0.196	0.031

Table 8

		N	Sig.
Pair 1	RA cm BTC & RA cm ATC	40	0.000
Pair 2	LA cm BTC & LA cm ATC	40	0.001
Pair 3	LV sys BTC& LV sys ATC	40	0.0018
Pair 4	LV dias BTC & LV dias ATC	40	0.000
Pair 5	RV basal BTC & RV basal ATC	40	0.006
Pair 6	RV mid BTC& RV mid ATC	40	0.002
Pair 7	RV base-apex BTC & RV base-apex ATC	40	0.003

DISCUSSION

This study was undertaken to establish the concept of cardiovascular hemodynamics alteration in massive pleural effusion, which could also be a reason for breathlessness, in addition to ventilation perfusion mismatch.

Previous animal experiments and patient studies had proved the cardiac tamponade physiology in massive pleural effusion, but with only a few parameters analysed and with confounders like small pericardial effusion, as in study by Traylor et al, it appears, therefore justified to make an attempt to rule out all possible confounders and to invoke all possible parameters altered in cardiac tamponade, to bring this concept to limelight.

In the study, out of 40 patients who had pleural effusion greater than ¾ of a hemithorax, 32 were males and 8 were females. Bilateral pleural effusion was found in 3 patients and of the remaining, 7 had left sided pleural effusion and 30 had right sided pleural effusion. Regarding etiology, in 25 cases it was due to tuberculosis, in the remaining, 1 case had decompensated liver disease and rest 14 had lung malignancy. Traylor et al[34]had studied a total of 27 patients dividing them into 2 groups, one with pleural effusion greater than ½ of hemithorax and the other with pleural effusion less than half of a hemithoarax.

In our study, 50% of patients had elevated jugular venous pressure and 63% had clinical evidence of pulsus paradoxus, 100% of bilateral effusions showed both, in comparison with Traylor et.al who had shown 36% in a group with pleural effusion greater than 1 hemithorax.

In echo parameters, for cardiac tamponade, 15% of the patients had right atrial collapse (which included 3 patients with bilateral pleural effusion, 2 patients with right sided pleural effusion, and 1 patient with left sided pleural effusion). 85% of the patients had significant tricuspid flow velocity respiratory variations, 80% had significant mitral flow velocity respiratory variations, 100% had significant pulmonary artery respiratory flow variation, and 85% had significant aortic flow velocity respiratory variation, all of which came to normal values after thoracentesis with significant p value.

The amount of pleural fluid tapped ranged from 1750ml to 2500ml, after which a repeat chest x ray was taken to assure that the pleural effusion remained less than a ½ of a hemithorax and all the clinical and echo parameters measured again within 24 hours. No chest tubes were placed for patients during study period. In Traylor et al. the amount of pleural fluid ranged from 2,000 to 4,000 ml (mean 3,050).

In Traylor et al. both groups, one with pleural effusion greater than 1 hemithorax, and the other with effusion less than a hemithorax, some patients, in addition, had small pericardial effusion, which could act as a confounder. This cofounder was eliminated in this study right from beginning by excluding patients with, even mild pericardial effusion from the study.

In this study, we had included other parameters, which could possibly be affected by the pressure changes in the pleural cavity, pericardial cavity like

RA size,

RV size at basal, mid septal level, and from base to apex,

LA size,

LV systolic dimension and

LV diastolic dimension.

From tables 7 and 8 it is clear that the parameters of cardiac chamber size of both right and left side of the heart, had shown statistically significant alteration with massive pleural effusion, before and after thoracentesis, reflecting that pressure changes in pleural cavity due to massive pleural effusion are transmitted to cardiac chambers through pericardium space, generating respiratory flow velocity variations

On comparing, clinical parameters reflecting hemodynamic status like $SpO_{2,}$ pulse rate, systolic blood pressure and diastolic blood pressure, before and after thoracentesis of massive pleural effusion in the study there is a statistically significant alteration in pulse rate, systolic blood pressure and diastolic blood pressure.

Regarding sidedness of pleural effusion, and clinically detected evidence of cardiac tamponade like elevated jugular venous pulse and pulsus paradoxus, in our study, no statistically significant correlation was found and any conclusion regarding sidedness of pleural effusion causing cardiac tamponade requires an invasive study, measuring and correlating intra-pleural, intra-pericardial pressure and cardiac chamber pressure to find out a plausible mechanism, to be carried out in a large population sample.

Even those patients who had massive pleural effusion without showing tricuspid and mitral respiratory variation not amounting to cardiac tamponade had their values well above the normal. This could be explained by the fact that there are significant interindividual variation in the size of heart, lungs, pleural cavities with their surrounding pleura, which determines the closeness contact of the pleura and pericardium , which in turn determines the amount of area of compression on the heart when the pleura is distended with pathological fluid collection. Moreover unlike pneumothorax which uniformly increases pleural pressure, pleural effusion creates a gradation in pleural pressure[6, 7], due to the dependency based distribution of fluid as well as the consistency of accumulating fluid.

This concept can be made clearer still, by further studies with invasive measurements correlating intra-pleural pressure, intra-pericardial pressure and cardiac chamber and great arteries pressure.

LIMITATIONS OF THE STUDY

1. Patients, who had significant respiratory flow variations across valves, yet, not amounting to tamponade features clinically, could not be explained clearly without invasive monitoring of pleural, pericardial and cardiac chamber pressures.
2. The exact location and distribution of pleural effusion cannot be ascertained only from chest x ray film, and needs a CT chest, which could explain why some patients with relatively lesser quantity of pleural effusion demonstrate remarkable tamponade physiology, whereas, some with larger quantities does not, was not planned in this study.
3. Because of the small study group, effect of sidedness of pleural effusion on cardiovascular hemodynamic alteration, could not be commented with statistical significance.

CONCLUSION

1. This study establishes that massive pleural effusion can have significant hemodynamic impairment as evidenced by altered Doppler AV filling profile.
2. Regarding sidedness of pleural effusion, there is no significant correlation between side of massive pleural effusion and development of cardiac tamponade physiology in this study.
3. There was no elevation of pulmonary arterial pressure in spite of mechanical effect of pleural effusion over the lung vasculature.
4. We believe that one of the mechanisms of dyspnoea in massive pleural effusion could be related to the compromise in cardiac hemodynamics.

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APPENDIX

PROFORMA

Age	
Male	
Female	
Clinical parameters	
Systolic blood pressure	
Diastolic blood pressure	
Elevated JVP	
Pulsus paradoxus	
Pulse rate	
Respiratory rate	
Spo2	
Co-morbid conditions	
Diabetes	
Systemic hypertension	
Renal failure	
Tuberculosis	
Malignancies	
HIV	
Heart failure	
Constrictive pericarditis	
Pericardial effusion	

ECHO PARAMETERS

Parameter	Before Thoracentesis	After Thoracentesis
Mitral A inspiration		
Mitral A expiration		
Mitral E inspiration		
Mitral E expiration		
Tricuspid A inspiration		
Tricuspid A expiration		
Tricuspid E inspiration		
Tricuspid E expiration		
Pulmonary flow velocity inspiration		
Pulmonary flow velocity expiration		
Aortic flow velocity Inspiration		
Aortic flow velocity expiration		
RA size		
LA size		
RV size base		
RV size at mid-septal level		
RV size base to apex		
LV systolic dimension		
LV diastolic dimension		

MASTER CHART

BEFORE THORACENTESIS																													
							DIMENSIONS							FLOW PARAMETERS																
														MITRAL INFLOW (cm/s)				TRICUSPID INFLOW (cm/s)				PUL ART FLOW		AORTIC FLOW				
#	AGE	SEX	CAUSE	SIDE	JVP	PARAODXUS	RA(CM)	LA(CM)	LV SYS	LV DIAS	RV BASAL	MID	BASE-APEX	E(INSP)	E(EXP)	A(INSP)	A(EXP)	E(INSP)	E(EXP)	A(INSP)	A(EXP)	INSP	EXP	INSP	EXP	SPO2	PR	SYS BP	DIA BP
1	45	M	TB	L	Y	Y	3.2	3.6	3	5.2	2.2	2.9	7.2	54	69	50	63	90	61	88	59	88	72	81	93	96%	102	100	68
2	53	M	TB	R	N	N	3.4	3.7	2.9	4.8	2.6	3	7.6	60	78	56	71	69	47	68	46	102	84	118	135	96%	111	120	88
3	43	M	MALIG	R	Y	Y	3	3.1	3.3	5.5	2.7	3	7.1	70	89	65	80	85	60	70	49	92	78	76	90	97%	118	90	60
4	47	M	MALIG	R	N	N	3.1	3.3	3	5.1	2.2	2.7	7.4	56	70	54	67	82	56	70	49	98	82	56	70	95%	98	100	70
5	32	F	TB	R	Y	Y	3.8	3.7	2.7	4.8	2.6	2.9	7.2	60	80	46	60	81	56	58	42	81	64	75	63	97%	102	110	70
6	47	M	TB	R	N	N	3.4	3.6	3.1	5	2	2.7	7.1	59	72	54	69	65	46	47	35	79	63	60	76	99%	100	100	78
7	39	M	TB	R	Y	Y	3.1	3.5	3.3	5.3	2.3	3.1	7.6	44	59	60	73	69	49	62	46	90	72	80	68	98%	97	110	80
8	57	M	TB	R	N	N	3.5	3.6	3.2	5.1	2.6	3.1	7.4	54	69	51	64	72	51	46	32	106	90	52	66	99%	103	120	78
9	59	M	MALIG	R	N	Y	3.5	3.7	3.5	5.4	2.7	3	7.3	56	72	63	81	80	60	78	69	80	59	67	83	97%	100	114	80
10	65	M	TB	L	Y	Y	3.2	3.1	3	5.1	2.8	3.1	7.3	57	70	58	72	76	58	74	55	74	65	74	90	98%	96	112	80
11	45	M	MALIG	R	N	N	3.6	3.6	2.9	4.9	2.2	2.9	7.8	58	72	65	85	69	48	72	52	75	60	70	87	97%	98	110	70
12	37	M	MALIG	R	N	N	3.7	3.9	3.1	5	2.1	2.7	7.1	54	69	67	81	73	51	68	48	72	59	64	78	98%	104	110	80
13	56	M	TB	R	Y	Y	3.6	3.7	3.4	5.4	2.1	2.8	7.6	52	68	63	79	68	47	55	39	68	56	74	90	97%	101	114	82
14	67	M	TB	R	Y	Y	3.8	3.7	3	5.1	2.4	3	7.5	50	63	60	73	68	50	61	45	92	76	81	97	99%	104	118	78
15	47	M	TB	R	N	N	3.4	3.3	2.8	4.9	2.3	3.1	7.4	60	73	66	78	82	64	52	40	100	85	84	98	96%	98	120	80
16	49	M	TB	L	Y	Y	3.6	3.9	3.2	5.1	2.6	3.3	7.4	56	75	66	81	78	55	70	48	90	75	75	89	95%	105	90	64
17	43	M	TB	R	N	Y	3.1	3.2	3.1	5	2.3	3.3	7.3	60	73	63	76	72	55	65	50	85	71	78	96	98%	109	108	70
18	54	F	TB	R	Y	Y	3.5	3.7	3	4.9	2.1	3	7.1	50	65	59	76	79	56	72	53	76	63	74	92	97%	103	120	78

19	56	F	MALIG	R	Y	Y	3.5	3.2	2.7	5	2.2	2.9	7.3	69	91	64	85	78	54	56	40	90	72	36	45	99%	102	128	86
20	50	M	TB	L	Y	Y	3.4	3.7	2.9	4.9	2.6	2.9	7.8	61	79	57	73	68	47	70	48	98	83	96	109	98%	99	104	78
21	45	M	TB	R	N	N	3.1	3.3	3	5.5	2.8	2.7	7.1	52	44	50	41	63	49	60	47	94	86	90	97	97%	98	108	76
22	46	F	TB	R	N	Y	3.7	3.1	3.1	4.9	2.1	3	7.5	46	60	40	54	51	35	47	33	81	68	58	72	96%	100	110	76
23	40	M	TB	L	N	N	3.2	3.9	3	5.1	2.3	2.8	7.2	57	71	55	68	83	57	71	50	100	83	57	72	98%	101	110	70
24	38	M	TB	R	N	Y	3.4	3.6	2.7	5	2.7	3	7.7	59	70	44	65	73	50	78	55	95	63	83	97	95%	102	100	60
25	30	M	MALIG	B	Y	Y	3.6	3.2	3.4	5.2	2.6	3.2	7.6	86	110	51	68	76	51	55	35	92	89	75	91	97%	114	120	78
26	42	F	TB	R	N	N	3.3	3.5	3.1	4.8	2.1	2.7	7.4	47	71	74	89	80	57	62	30	75	42	65	85	96%	98	116	68
27	26	M	TB	L	Y	Y	3.5	3.4	2.9	5.3	2.4	2.4	7.4	33	43	59	77	78	54	56	35	79	43	77	91	97%	96	110	80
28	43	M	TB	R	Y	Y	3.7	3.5	3.3	5.4	2.3	3.1	7.3	45	60	49	69	68	48	72	52	68	35	69	87	98%	100	118	78
29	38	F	MALIG	R	N	N	3.4	3.3	2.9	5.1	2.8	2.9	7.1	47	73	66	74	72	50	62	33	64	32	87	101	96%	102	100	72
30	60	M	MALIG	B	Y	Y	3.2	3.7	3.5	5	2.3	2.7	7.1	54	74	63	70	79	53	46	20	69	35	70	91	95%	101	96	68
31	45	M	MALIG	R	N	N	3.5	3.5	3.6	4.8	2.5	2.5	7.6	52	73	51	66	65	40	81	40	75	40	59	75	95%	99	102	74
32	75	M	MALIG	R	N	N	3.6	3.7	3.2	5.2	2.7	3.1	7.2	61	97	59	77	82	57	79	37	72	35	45	67	95%	105	104	86
33	56	M	TB	R	N	N	3.1	3.5	3.4	5.2	2.1	3.2	7.7	50	74	67	80	76	40	70	51	77	37	85	99	99%	108	110	82
34	38	M	TB	R	Y	Y	3.4	3.2	3	5.4	2.5	2.7	7.1	35	45	36	56	83	56	58	35	101	60	63	84	96%	99	124	74
35	40	M	TB	R	Y	Y	3.3	3.3	2.6	5.1	2.3	2.9	7.5	49	73	58	80	82	53	46	20	95	58	49	70	97%	98	126	66
36	37	M	TB	L	Y	Y	3.7	3.4	2.9	5.5	2.6	3	7.4	46	65	60	72	65	40	65	31	92	55	67	85	95%	102	116	60
37	42	M	TB	R	Y	Y	3.2	3.6	3.2	4.8	2.1	2.8	7.3	33	46	57	78	79	53	53	34	85	50	78	97	98%	97	118	78
38	50	M	TB	R	N	N	3.2	3.7	3.3	5	2.2	3.3	7.4	51	74	69	80	74	50	58	39	81	49	76	93	96%	103	106	80
39	26	F	DCLD	B	Y	Y	3.6	3.6	3.5	5.4	2.4	3.1	7.2	56	78	53	64	75	49	64	45	100	78	56	75	97%	99	104	70
40	56	M	TB	R	N	Y	3.5	3.3	3	5.2	2.3	2.7	7.7	47	72	64	82	71	50	66	46	95	64	87	104	95%	98	112	86

AFTER THORACENTESIS																											
					DIMENSIONS							FLOW PARAMETERS															
												MITRAL INFLOW (cm/s)				TRICUSPID INFLOW (cm/s)				PUL ART FLOW		AORTIC FLOW					
#	AGE	SEX	JVP	PARAODXUS	RA(CM)	LA(CM)	LV SYS	LV DIAS	RV BASAL	MID	BASE-APEX	E(INSP)	E(EXP)	A(INSP)	A(EXP)	E(INSP)	E(EXP)	A(INSP)	A(EXP)	INSP	EXP	INSP	EXP	SPO2	PR	SYS BP	DIA BP
1	45	M	N	N	3.1	3.6	3.1	5.4	2.3	2.9	7.3	45	54	67	76	80	67	97	78	90	81	86	92	97%	90	104	78
2	53	M	N	N	3.4	3.7	2.8	4.9	2.5	3	7.5	84	95	65	76	60	51	58	44	98	86	111	120	98%	95	120	86
3	43	M	N	N	3.1	3.1	3.4	5.5	2.9	3.1	7	66	80	62	76	72	60	70	58	86	74	80	92	98%	98	100	70
4	47	M	N	N	3.3	3.2	3.3	5.4	2.4	2.8	7.5	56	65	51	62	78	66	67	55	102	91	55	63	97%	97	110	74
5	32	F	N	N	3.6	3.3	2.6	5	2.6	2.9	7.1	70	82	42	50	75	60	58	49	78	69	72	64	97%	98	110	80
6	47	M	N	N	3.2	3.6	3	5.1	2.2	2.6	7.2	35	41	55	66	63	56	47	37	80	69	66	75	98%	96	110	80
7	39	M	N	N	3.1	3.4	3.3	5.3	2.2	3.3	7.7	46	55	64	72	60	45	57	42	85	72	42	36	98%	100	116	80
8	57	M	N	N	3.4	3.5	3	5.2	2.5	3.1	7.2	53	65	50	60	70	54	44	34	96	80	50	60	98%	92	110	78
9	59	M	N	N	3.3	3.5	3.4	5.3	2.6	3	7.3	55	68	66	79	78	60	78	61	76	66	70	81	99%	93	120	80
10	65	M	N	N	3.1	3	3.1	5.1	2.7	3	7.2	59	68	60	71	72	60	74	59	70	61	75	88	100%	93	120	82
11	45	M	N	N	3.5	3.6	2.7	5	2.3	2.8	7.8	55	68	66	80	70	55	70	58	73	65	73	85	99%	96	110	80
12	37	M	N	N	3.7	3.6	3	5.3	2.2	2.6	7.2	54	65	69	78	66	50	65	50	68	58	63	75	98%	93	120	76
13	56	M	N	N	3.5	3.7	3.9	5.3	2.3	2.7	7.6	54	65	61	73	66	50	51	41	66	58	72	86	97%	97	110	86
14	67	M	N	N	3.6	3.6	3.1	5.2	2.4	3	7.6	54	61	63	71	69	56	62	51	86	73	82	93	98%	90	124	80
15	47	M	N	N	3.3	3.2	2.7	5	2.4	3.1	7.4	60	70	67	76	73	56	63	47	96	88	85	96	98%	90	120	80
16	49	M	N	N	3.5	3.8	3	5.1	2.5	3.4	7.5	57	69	71	80	70	56	60	48	88	77	76	85	99%	90	126	78
17	43	M	N	N	3.2	3	3	5.2	2.3	3.2	7.4	56	66	59	72	66	51	78	66	81	73	80	92	98%	92	120	80
18	54	F	N	N	3.5	3.6	3.1	5	2.2	3.1	7.3	53	63	60	74	72	61	66	54	73	65	74	87	99%	90	118	78
19	56	F	N	N	3.5	3.2	3.5	5.1	2.3	3	7.5	62	76	61	75	69	56	52	44	92	79	44	52	99%	91	118	80
20	50	M	N	N	3.3	3.4	3.2	5.1	2.5	2.9	7.8	58	71	55	68	63	51	66	52	96	87	98	107	99%	89	120	80
21	45	M	N	N	3.1	3.3	3.4	5.4	2.9	2.8	7.3	50	43	47	39	60	50	58	49	96	88	91	99	98%	92	110	80

22	46	F	N	N	3.4	3	3	5.1	2.2	3	7.6	50	59	46	53	50	41	45	39	87	76	60	70	99%	90	116	82
23	40	M	N	N	3.1	3.7	3.1	5.2	2.3	2.9	7.5	55	64	52	62	76	59	65	55	98	86	68	76	99%	88	120	84
24	38	M	N	N	3.3	3.5	3.8	5.1	2.8	3.1	7.8	59	67	53	68	66	48	69	48	69	57	45	54	99%	92	110	70
25	30	M	N	N	3.5	3.2	3.4	5.3	2.7	3.2	7.6	47	66	67	80	40	28	68	46	88	75	67	78	98%	97	124	80
26	42	F	N	N	3.2	3.4	3.1	5	2.2	2.9	7.6	49	65	59	70	72	50	60	45	83	70	73	84	97%	87	116	80
27	26	M	N	N	3.4	3.4	3.5	5.3	2.2	2.5	7.4	45	54	60	74	69	45	51	34	86	74	45	56	99%	98	112	80
28	43	M	N	N	3.5	3.5	3.5	5.3	2.4	3	7.5	65	79	47	64	72	54	78	59	68	56	47	60	98%	100	120	80
29	38	F	N	N	3.3	3.3	3.2	5.1	2.8	3.1	7.2	45	57	62	77	69	44	49	32	66	56	68	75	99%	97	104	74
30	60	M	N	N	3.2	3.6	3.6	5.1	2.4	2.8	7.4	56	69	60	75	65	40	67	50	64	50	71	83	98%	95	100	70
31	45	M	N	N	3.3	3.5	3.7	4.9	2.7	2.6	7.6	46	54	65	79	80	58	44	30	63	52	63	75	97%	97	102	74
32	75	M	N	N	3.5	3.5	3.6	5.3	2.7	3.2	7.3	34	40	39	55	78	57	48	34	96	84	84	93	98%	99	104	86
33	56	M	N	N	3.1	3.5	3.8	5.2	2.3	3.2	7.7	56	67	48	66	73	55	43	25	91	75	76	85	98%	92	110	80
34	38	M	N	N	3.3	3.2	3.4	5.4	2.4	2.9	7.3	59	68	61	72	72	52	57	40	68	52	63	75	98%	91	130	76
35	40	M	N	N	3.1	3.2	3.6	5.2	2.4	3	7.5	66	86	71	83	65	47	54	34	85	70	79	90	99%	89	126	70
36	37	M	N	N	3.5	3.4	3.1	5.5	2.6	3.1	7.5	55	69	59	74	68	47	65	49	90	75	48	57	99%	92	120	70
37	42	M	N	N	3.1	3.6	3.5	5.1	2.4	3	7.4	54	61	69	84	75	57	70	52	65	51	45	55	96%	92	120	80
38	50	M	N	N	3.2	3.6	3.6	5.1	2.3	3.3	7.4	50	63	52	67	72	50	73	55	75	55	78	90	97%	95	110	80
39	26	F	N	N	3.5	3.6	3.2	5.4	2.4	3.2	7.4	56	68	57	74	62	45	75	58	72	60	83	94	96%	90	104	74
40	56	M	N	N	3.4	3.3	3.1	5.3	2.4	2.9	7.7	53	66	45	62	60	41	53	37	69	52	91	102	99%	94	112	88